ALCOHOL INTOXICATION AND EMOTION

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I hereby declare that this thesis is the result of research and composition solely by myself.

M. Muñoz

To my parents.

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During this time there were friends who always believed, more than myself, that I was going to succeed. With them I will always be in debt.

ABSTRACT

This thesis represents an attempt to study the effects of alcohol intoxication on emotional behaviour in normal individuals.

The pharmacology of ethanol was reviewed briefly and a framework for the study of emotion and the effects of drinking on it was delineated within the general information processing paradigm. The experimental research carried out in this thesis concentrated on two aspects of emotional behaviour that have particularly attracted the interest of those investigating the effects of alcohol: sexual response and anxiety.

In an experiment that used the Balanced Placebo Design alcohol was found to reduce the ability to voluntarily inhibit sexual response to erotic stimulation. The most likely explanation of this effect is an impairment of the cognitive processes necessary to supress the physiological sexual reaction.

A review of the literature reveals that a relatively high dose of alcohol (more than 0.8 g/kg) consistently reduces cardiac response to social stress. The effect of a lower dose was investigated. A repeated measures design was used in order to minimize the error variance due to the inter-individual variability in the effects of alcohol that it was thought had obscured the effect of low doses in previous studies. Despite the fact that the response to the social stress task habituated from the first to the second session, which complicated the results, this study provided some support to the thesis of the anxiety reducing effects of moderate doses of alcohol.

In order to investigate the effects of alcohol on the primary response to stressing environmental stimuli, the psychophysiological response to high-intensity auditory stimulation was examined. Alcohol seemed to make subjects react to the stimuli in a more defensive, less receptive manner. This effect might have been caused by the blood alcohol concentration still increasing at the moment of the test.

It has been argued that the effects of alcohol consumption on affect are the indirect result of the action of ethanol on 'cognitive-perceptual processing'. An experiment was conducted which investigated the effects of alcohol on the perception of emotional expression. Alcohol impaired perception of facial expression of emotion but it did not affect judgement of oral expression of emotion.

The results of these experiments point in general to a pharmacologically determined impairment of information processing functions, which might influence emotion. The so-called cognitive explanation of the effects of alcohol on emotion is discussed critically. The methodological problems, in particular design and placebo manipulation procedures, of the experimental study of the acute effects of alcohol are also discussed.

Contents

Chapter 1.

BASES OF A PSYCHOPHARMACOLOGY OF POSTDRINKING EMOTIONAL BEHAVIOUR

1.1. Introduction

The present thesis is concerned with alcohol intoxication, which has been defined as "alterations in mental processes and behaviour that sometimes accompany moderate to heavy drinking on a single occasion" (Chick, 1984, p. 47). We are interested in the short-term effects of ethanol on the psychological subject. The question to be addressed is as follows: how is psychological functioning affected by the fact that a certain dose of alcohol has been consumed? Emotion will be the central subject of concern. Thus, the purpose of this thesis will be to study how ethanol modifies mood, feelings and emotional experience and behaviour when consumed in a socially accepted amount by people not thought of as problem drinkers. Neither the chronic effects of alcohol nor problem drinking will be examined directly.

Alcohol intoxication is worth studying in its own rights. Acute outcome of alcohol use is the cause of a number of important social and personal hazards and troubles: alcohol plays a significant role in many kinds of accidents, and in particular road accidents, public and family violence, sexual abuse, etc., whether the subjects of these actions are addicts or not. On the other hand, both folk wisdom and scientific models, from the early ones (e.g. Jellineck, 1960) to the most recent ones (e.g. Robertson et al., 1984), assume that alcohol intake yields some 'reinforcing consequences' which motivate people to drink, and it is obvious that people engage in drinking because of the short-term

effects of alcohol. Understanding the nature of the acute effects of alcohol will therefore be a preliminary to understanding chronic alcohol abuse. Moreover, it has been suggested that the chronic consequences of alcohol abuse on cognitive functioning are similar to those observed immediately after consumption (Robertson, 1985).

1.2. Pharmacological properties of alcohol

It seems reasonable to expect that the answer to the question 'how does alcohol affect behaviour?' will lie in the physiological action of alcohol. After all, the behavioural consequences of drinking are due to the fact that the Central Nervous System (CNS) is powerfully affected by alcohol. In what follows I will summarize the pharmacology of alcohol trying to give a brief account of those aspects of alcohol pharmacokinetics and of ethanol action on the CNS that might be relevant to explaining intoxicated behaviour.

1.2.1. Absorption, distribution and metabolism of ethyl alcohol

The absorption of ethanol takes place mainly in the stomach by the simple process of diffusion. Some ethanol may reach the intestine where it is absorbed at a faster rate than in the stomach. The rate of absorption depends on the alcohol concentration of the drink and the amount of food present in the stomach. Food present in the stomach will delay stomach emptying, so that less alcohol reaches the intestine. The more concentrated the alcoholic beverage is, and the less food there is in the stomach, the faster the absorption is. The rate of absorption determines the blood alcohol level reached with a certain dose. The presence of food in the stomach will also influence the total

amount of alcohol that reaches the brain. As the rate of elimination is constant, and because the alcohol passes through the liver where it is metabolized before it reaches the brain, a slower absorption rate will mean that some ethanol never reaches the brain. Understandably, the rate at which alcohol is consumed will also influence the rate of entry in the blood.

Ethanol does not dissolve well in fat but it easily diffuses through the body water, and has no problems in crossing the blood-brain barrier. Concentrations of alcohol in body water are about ten times higher than in body fat (Goldstein, 1983).

Most ethanol (90-95%) that gets into the blood is metabolized in the liver, the rest is excreted unchanged in the breath and the urine. The metabolism of alcohol in the liver is a process of oxidation to acetaldehyde. This can be carried out through various enzymatic pathways, but the alcohol dehydrogenase is the main route. The alcohol dehydrogenase utilizes nicotinamide adenine dinucleotide (NAD) as coenzyme to convert ethanol to acetaldehyde.

$$CH_3\ CH_2\ OH + NAD^+ \longrightarrow CH_3\ CHO + NADH + H^+$$

Most of the acetaldehyde formed is metabolized in the liver to acetate, a substance with little if any toxicity. Although small amounts of acetaldehyde get into the blood stream, it does not seem to pass across the blood-brain barrier. Acetaldehyde is quite a toxic compound but it does not appear to contribute to the effects of alcohol consumption. Only if the elimination of the acetaldehyde is prevented (e.g. by the administration of disulfiram), thereby provoking its accumulation, does

the pharmacological action of acetaldehyde become obvious, via various unpleasant sympathomimetic symptoms (Goldstein, 1983).

Thus, ethanol itself and not its metabolites is the primary causal agent for the effects observed after the consumption of alcoholic beverages.

1.2.2. Biochemical effects of ethanol on the CNS

It has been known for some time that the primary site of the action of ethanol in the CNS is the neuronal membrane (Grenell, 1972). Ethanol can be thought of as an anaesthetic drug, and as such it exerts a general depressant effect on the CNS. Like most anaesthetics, ethyl alcohol has a general effect of a physico-chemical nature on the cell membrane (Ryall, 1989). It dissolves in the membrane lipids and causes membranal disorganization, which disrupts the function of the membrane proteins, and consequently alters the functioning of the membrane and the synaptic transmission processes (Goldstein, 1983). The effect of ethanol on the presynaptic membrane is that of inhibition of the release of the neurotransmitter; at the postsynaptic site the action of alcohol is more complex and it cannot be described in general as inhibitory or excitatory (Littleton, 1985).

After a comprehensive review, Hoffman and Tabakoff (1986) summarize the present knowledge on the effects of alcohol on brain biochemistry as follows: (a) "ethanol can affect the metabolism and function of nearly every neurotransmitter that has been studied"; (b) the acute action of ethanol on many processes seems to be biphasic; and (c) "ethanol may selectively affect neurotransmitter metabolism in a particular brain region or neuronal pathway" (Hoffman and Tabakoff,

1986, p. 47). They emphasize that "it is becoming clear that one must understand the complex interactions of the neurotransmitter systems in brain [...] in order to understand the response to ethanol" (p. 47). This point is perfectly illustrated in the study of the effects of ethanol on the noradrenaline (NA) system. Although ethanol has only a depressant effect on NA neurons as demonstrated by electrophysiologic studies, low doses of alcohol cause an increase in NA turnover. This can only be explained in terms of the action of ethanol on other neurotransmitter systems which act on NA neurons.

In sum, ethanol exerts a nonspecific effect on neuronal membranes that may result in specific effects on the different neurotransmitter systems. The scope of ethanol's action, which affects every nerve cell, and the complexity of the interaction amongst neurotransmitter systems (and our present limited understanding of it), makes it extremely difficult to complete the picture of the action of ethanol on the brain at the biochemical level. Moreover, the relationship between different neurophysiological actions of ethanol and behavioural effects remains to be established.

The interaction of ethanol with the gamma-Amino-butyric acid (GABA) system has attracted special mention. GABA is the major inhibitory neurotransmitter system in the brain. It mediates both presynaptic and postsynaptic inhibition of neuronal activity. GABA mediated neurotransmission may account for a third of all synapses in the mammalian brain (Iversen, 1982). It has been known for some time now (Hoffman and Tabakoff, 1986) that ethanol increases GABA mediated inhibition. Recently, it has been found that the administration of a benzodiazepine (the imidazobezodiazepine RO 15-4513) to rats

prior to the administration of ethanol reduces alcoholic intoxication, as rated on a scale which took into account exclusively signs of intoxication that involved the motor system, i.e. reduced muscle tone, motor incoordination, etc. (Suzdak et al., 1986). It seems that alcohol acts on benzodiazepine recognition sites that interact with the GABA-A receptors and that this GABA mediated physiological action of alcohol accounts for at least the motor aspects of alcoholic intoxication in rats.

1.2.3. The concept of disinhibition and the neurophysiology of ethanol intoxication

The 'hypothesis of disinhibition' has dominated lay views about alcohol as well as professional approaches and much of the research done up to the present day. The concept of disinhibition has a formulation within the realm of physiology that has survived for more than a century and we can find it openly expressed in a modern reference textbook of pharmacology: "alcohol, like other general anaesthetics, is a primary and continuous depressant of the CNS. The apparent stimulation results from the unrestrained activity of various parts of the brain that have been freed from inhibition as a result of the depression of inhibitory control mechanisms" (Ritchie, 1985, p. 374).

This conception can be traced back at least to the work of J.H. Jackson, the 'father' of British neurology, and his theory of 'evolution and dissolution' of the nervous system. Jackson's model of the CNS, which was very much inspired by Spencer's theory of evolution, was an attempt to account for a great deal of observations of the outcome of brain damage, experimental brain transections, and the administration of diverse drugs -particularly anaesthetics-, which had been

accumulated for decades. For Jackson, the higher parts of the brain have evolved from the lower zones. These are more organized than the higher regions, whose organization at birth has yet to be accomplished through processes like learning. Lower regions are the control site for automatic primitive behaviour and are subjected to the inhibitory control of higher regions. Ablation, damage or drug induced depression of the higher levels would cause 'over-activity' or 'unrestrained activity' of the lower areas. Thus, Jackson explained the behavioural effects of alcoholic intoxication in a similar way to Ritchie: "I submit that the uproarious drunkard is a duplex mental state with correspondingly double physical states -that more or less of the highest ranges of his highest cerebral centres is out of function; his uproariousness is the outcome of over-activity of the lower ranges of those centres which have not yet succumbed to the poison" (Selected writings of John Hughlings Jackson, edited by J. Taylor, 1958, vol. 2, p. 418).

Is there any modern evidence that supports Jackson's model and explains its survival until today? Electrophysiological studies have shown that the depressant effects of alcohol on the different regions of the CNS depend on synaptic density. Thus, the association cortex and, in second place, the reticular formation, whose structures involve a great number of synapses, are the most sensitive areas to the action of ethanol. The action of ethanol extends down to lower regions of the CNS only if the dose is increased (Himwich and Callison, 1972). This supports 19th century ideas.

It seems then that the 'higher regions', association cortex, are the most easily affected by alcohol, but is there any evidence that the association cortex exerts inhibitory control over the lower parts of the brain? There

are data suggesting that the cortex as a whole exerts inhibitory control over emotional impulses. In a classic work, Bard and Mountcastle (1948, cit. in Frijda, 1986) observed that dogs and cats in whom the entire cortex had been removed showed violent sham rage to slight provocations. In modern neuropsychology we also find evidence of the inhibitory control that the cortex exerts on lower parts of the brain. It has been shown (Tucker, 1986) that patients with frontal damage (in particular in the right hemisphere) show what has been called 'disinhibition syndrome': "the patients are inappropriately impulsive in social settings, and make crude jokes and sexual advances" (Tucker, 1986, p. 280). Emotional impulses and fixed unlearned behavioural patterns of emotional and motivational nature can be elicited by direct stimulation of lower brain stem sites. The control that different upper brain structures exert upon the lower parts seems to be of a dual nature. Both the amygdala and the frontal lobe - the hypothalamus could also be included here- contain "antagonistic, reciprocally acting divisions", which influence lower structures and behaviour in opposite directions. Thus, for example, while orbitofrontal lesions produce affective disinhibition, lateral damage disrupts planning and generates apathy (Frijda, 1986).

1.2.4. The physiological explanation of the behavioural effects of acute consumption of alcohol

From biochemical studies, recent findings on the interaction between ethanol and the GABA look promising, but could alcohol action on the GABA neurons explain alcohol intoxication? This question cannot be answered if previously what alcohol intoxication involves has not been specified. Suzdak et al (1988) equate alcohol intoxication and motor impairment. This is an example of a mistake that should be avoided.

Specific neurochemical effects of alcohol may probably account for different behavioural aspects of intoxication, but it seems unlikely that the effects of a drug that affects every neuron could be explained by its action on a single system of neurotransmission. To what extent or which aspects of alcohol intoxication can be explained by this single mechanism is an empirical question. I agree with Oatley (1978) that "we only understand physiological mechanisms [underlying behaviour] in so far as we have good psychological theories, i.e. theories of the logical structure of behaviour" (p. 7). Without a good description at the psychological level of what alcohol intoxication implies it would be impossible to ascertain the physiological mechanisms that underlie the behavioural effects of drinking. The psychology of alcohol intoxication should not be a list of symptoms but it should look at how psychological processes (that is, the processes by which the subject perceives, interprets and responds to the environment) are altered after the consumption of alcohol.

The attempt to equate alcohol intoxication with the impulsive behaviour shown in frontal lesions or with the easily triggerable rage of cats and dogs after the extirpation of the cortex reveals a legitimate (and recommendable in science) search for similarities between phenomena, but also a tendency, deeply rooted in our culture, to seek for tangible referents of not easily apprehensible facts. At most one could accept that there may be in alcohol intoxication some components of a lost cortical inhibitory function, but, before we can grant more credibility to these neuroanatomical hypotheses, it should be established beyond superficial description of behaviour what the precise similarities are between alcohol intoxication and the outcome of these cortical lesions.

It is interesting to observe that a circular explanation is often used. The hypothesis of an inhibitory cortical control upon lower structures is used to explain the effects of alcohol on behaviour (e.g. Jackson, 1958, Ritchie, 1985); and the behavioural disinhibition observed after drinking is used as a proof of the cortical inhibitory control (e.g. Frijda, 1986).

1.3. Psychology of the short-term effects of drinking

The need for explanatory theories of postdrinking behaviour at the psychological level is emphasized by the evidence that psychological - nonpharmacological- factors play a major role in shaping human behaviour after drinking. An important part of this evidence stems from a number of experiments in which the experimental design known as 'balanced placebo' design (BPD) has been employed. The main finding of this line of research, which has dominated psychological research on alcohol for at least fifteen years, is that the mere belief of having consumed an alcoholic beverage is enough to make people feel and behave in the manner that we expect when they have consumed alcohol. On the other hand, anthropological data also suggest a crucial role for nonpharmacological cultural factors in determining postdrinking human behaviour. As an example of the paradoxical effects of ethanol in different cultures Chick (1984) mentions the cases of a Micronesian culture and the Camba Indians of Bolivia. While alcohol produces extremely destructive and aggressive behaviour in the members of the Micronesian culture, the Bolivian Indians become incredibly quiet and peaceful after drinking. This radical difference in the effects of alcohol cannot be explained on the grounds of any physiological mechanism.

1.3.1. BPD research

The BPD was used to study the effects of alcohol for the first time in the early seventies (Engle and Williams, 1972; Marlatt, Deming and Reid, 1973). The BPD is a 2 x 2 factorial design in which information about the content of the drink and the actual content of the beverage are manipulated independently (see fig 1.1). This results in four experimental groups formed by (1) subjects who are told that their drinks contain alcohol but are given a soft drink; (2) subjects told that their drinks contain alcohol and who actually receive an alcoholic beverage; (3) subjects told that their drinks do not contain any alcohol and who are given soft drinks; and (4) subjects told that their drinks do not contain any alcohol but who are given a beverage containing alcohol.

During the 1970's several research groups used this design to study the effects of drinking on a variety of behaviours: 'craving for alcohol', aggression, anxiety, mirth, sexual arousal and motor and cognitive abilities. These experiments showed that postdrinking behaviour, when of an emotional or social nature, was determined not by the physiological action of alcohol (that is, by the actual consumption of ethanol) but by the mere belief that alcohol had been consumed regardless of the actual content of the drink. That is, the instructional manipulation in the BPD was sufficient to alter behaviour. The typical finding of these studies was, at least in the beginning, that instructional manipulation was the only factor whose effects reached statistical significance; alcohol was, however, the main determinant of the detrimental effects on motor and cognitive performance after drinking

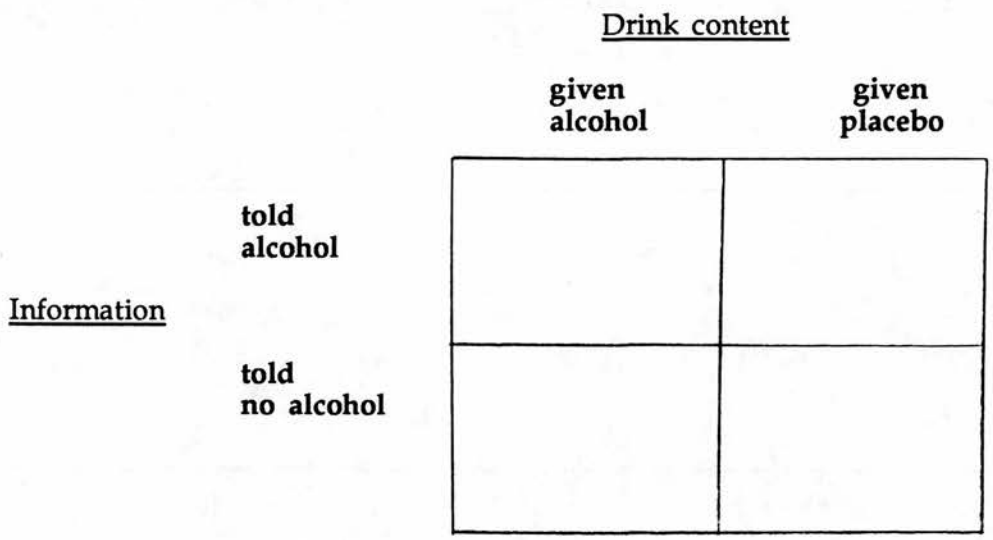

Fig 1.1. Balanced Placebo Design

(see Marlatt and Rohsenow ,1980, for a review of these early findings).

1.3.2. First assessment of the results of BPD studies

Marlatt and Rohsenow (1980) comprehensively reviewed the BPD research done in the 1970's and tried to find theoretical explanations for these findings. Their paper is a necessary point of reference from which to study how the views and interpretations of the results of this line of research have evolved.

But, first of all, it should be noted that the effect of the instructional manipulations has been, since the first BPD experiments, referred to as 'expectancy effect', assuming, implicitly in most cases, that expectancies concerning the effects of alcohol held by the subjects were the crucial variables responsible for the nonpharmacological effects of drinking. As Maisto, Connors and Vuchinich (1978) pointed out, as actual expectancies have not usually been assessed in these studies, the use of the expectancy construct to explain these results represents an interpretation going beyond the actual data. There is nothing wrong with theoretical interpretations, provided that one is aware of them. But this has not usually been the case here. Consequently, it is preferable to describe the results of BPD studies in a more neutral way as 'effects of the belief of having consumed alcohol' rather than as 'expectancy effects'.

In 1980, when Marlatt and Rohsenow published their review paper, it was the view of most researchers in this area that BPD experiments had proved that the effects of moderate drinking were the result of a psychological rather than a physiological process. Marlatt and

Rohsenow (1980) summarized and interpreted the results of the first eight years of research in this area in the following way:

> "... a tentative pattern of results emerges. Expectancy effects predominate over the pharmacological properties of alcohol both with consummatory behaviours (craving and beverage consumption) and with interpersonal/social behaviours for males including aggression, social anxiety, and sexual arousal. [...] At least for male subjects, the findings reported are in line with contemporary cultural beliefs and stereotypes about the effects of alcohol: that alcohol will serve to increase aggressive behaviour and sexual arousal, while at the same time reducing tension or anxiety. The variable findings obtained with females may reflect the ambivalent attitudes that society has adopted about women who drink and the effects of alcohol upon their behaviour. [...] Expectancy effects are decreased or absent altogether with selected responses such as reaction time or simple motor tracking skills (e.g. performance on the pursuit rotor). With these responses, alcohol itself has a deleterious effect, regardless of the expectancy manipulation." (Marlatt and Rohsenow, 1980, p. 185)

For Marlatt and Rohsenow (1980), socially acquired expectancies concerning the effects of alcohol are the main determinants of postdrinking behaviour both in BPD experiments and in real life. How can expectancies shape postdrinking behaviour? Marlatt and Rohsenow (1980) proposed Valins' extension (Valins,1970) of Schachter's attribution theory of emotion (Schachter, 1964) as an adequate model to explain BPD results (i.e. that instructional manipulation emerges as the only factor that significantly affects behaviour). Schachter's theory, derived

from a well-known pioneering study, states that given a state of physiological arousal, this state will generate 'evaluative needs', which will lead the individual to 'label' his state and his feelings 'in terms of the cognitions available to him'. 'Emotional states are a function of the interaction of such cognitive factors ['available cognitions'] with a state of physiological arousal' (p. 53). The process of physiological arousal is central in Schachter's theory. Later, Valins (1970) showed that a previous state of physiological arousal is not essential to generate an emotional state and that only the belief that one is being affected by emotional stimuli is sufficient to determine the emotional state and the subsequent emotional behaviour. As interaction effects between alcohol itself and expectancy have not normally been found using the BPD (that is, the mere belief of having drunk alcohol influenced behaviour regardless of whether alcohol was consumed or not) the expectancy effect (or, better, the belief or instructional effect) found in these studies fits Valins' conception rather than Schachter's theory.

1.3.3. Reassessment of BPD research

BPD research continued throughout the 1980's. Hull and Bond (1986) conducted a meta-analysis of studies investigating psychological and pharmacologically mediated effects of drinking that had used the BPD. The results of this meta-analysis showed that both alcohol consumption and experimental instructions had significant effects on behaviour. Additionally, both alcohol consumption and expectancy effects were associated with significant heterogeneity. The effect of alcohol was found to be twice as large as that of instructional manipulation. The statistically significant heterogeneity indicates that the effect of both alcohol and instructional manipulation vary across studies. The

dichotomy social-nonsocial behaviour explains part of the heterogeneity of the effect of instructional manipulation, in the sense that social behaviour tends to be more easily affected by placebo manipulation. Individual differences on various dimensions are tentatively proposed by Hull and Bond (1986) to account for the remaining heterogeneity.

Thus, Marlatt and Rohsenow's earlier conclusion that "expectancy effects predominate over the pharmacological properties of alcohol" (Marlatt and Rohsenow, 1980, p. 185) is not upheld. Actual drinking even in the moderate doses that BPD studies typically use (i.e. 0.4-0.6 g/kg of body weight) seems to affect behaviour more effectively than what suggested by the earlier BPD studies. It is my view that the contribution of BPD studies has been to demonstrate that placebo factors can affect postdrinking behaviour and, therefore, they have to be taken into account. Some interpretations, particularly in the beginning, tended to suggest that intoxicated behaviour was exclusively the result of the belief that one had drunk and the learned expectancies concerning alcohol effects. Even if that is true in the course of a BPD experiment, it does not necessary follow that it applies to real life drinking. There is evidence that the placebo effects induced by the instructional manipulation are very clear between 0 and 30 min after drinking, but after that the influence of the induced beliefs decreases (Sher, 1985). Further, placebo effects also tend to be stronger in a group setting (Sher, 1985).

BPD research has demonstrated that psychological processes may be important determinants of alcohol intoxication. In societies with a defined system of beliefs and expectancies concerning alcohol effects, these psychological factors must be operating on every drinking

occasion. Placebo effects outside the lab may be even stronger than in the lab. It is even possible to imagine that in some real life situations the duration of the placebo effect could be longer than 30 min. But this does not mean that alcohol itself does not have any actual pharmacological effect, nor even that the placebo effects 'predominate' over the pharmacological action. We could establish a parallel between alcohol and prescribed medicines. Is the fact that placebo factors are sometimes powerful curative agents a proof that prescribed medicines are inert substances? Alcohol is a potent drug with a wide range of actions on the CNS. Human beings have made use of its intoxicating effects for thousands of years in many different cultures and civilizations throughout history. It is unlikely that this would have occurred without a real physiological effect of alcohol. The pharmacological action of alcohol will no doubt interact with nonpharmacological factors (e.g. expectancies), but the nature and characteristics of this interaction cannot be ascertained if the actual effects of alcohol on psychological functioning are not previously understood. BPD research showed us that nonpharmacological factors should not be disregarded. Bearing this in mind, future research should aim to understand the pharmacological effects of alcohol on the psychological functioning.

1.4. Psychology of emotion

1.4.1. Nature and components of emotional phenomena

Since the concern of this thesis is the effect of alcohol on emotion, it is necessary to define emotion or at least to try to outline what is understood by emotion. This is not an easy task. Reber's Dictionary of

Psychology affirms that "historically this term [emotion] has proven utterly refractory to definitional efforts; probably no other term in psychology shares its nondefinability with its frequency of use" (Reber, 1985, p. 234).

For a long time theories of emotion have tended to focus on a particular aspect of emotional phenomena, disregarding any other facets except the one they favour. As Leventhal and Tomarken (1986) have pointed out, a number of theories have been put forward over the last century, each one concentrating on a partial aspect of emotion (e.g. emotions as discreet patterns of expressive response, emotions as the product of patterns of autonomic response, emotions as the activity of certain structures of the CNS, etc.). The standard account of the psychology of emotion is usually (e.g. Strongman, 1987) little more than a census of different theories grouped according to the aspect they highlight. But these different perspectives, if failing to provide a comprehensive model, have served to define the range of emotional phenomena and specify their features and components. Most theorists today would agree that emotional phenomena necessarily comprise (a) a physiological reaction, (b) a subjective experience, and, in most cases, (c) motor-expressive behaviour (Izard, Kagan and Zajonc, 1984). Also, it is usually accepted that emotions represent (or have represented at some point in the course of the evolution of the species) a way of interacting with certain environmental circumstances in an effective and economical manner. Emotions have developed in the course of evolution to 'decouple' the rigid stimulus-response associations of reflexes and 'instinctive innate releasing mechanisms' (Scherer, 1984). The idea that emotions have a biological basis and serve the purpose of adaptation is shared by most theorists today (e.g. Izard, 1984, Frijda,

1986, Buck, 1984).

Emotions are "action tendencies" (Izard, 1984) or "changes in action readiness" (Frijda, 1986) elicited by external or internal events which are significant to the individual. Emotions "result from the interaction of an event's actual or anticipated consequences and the subject's concerns" (Frijda, 1986, p. 6). Simple reflex mechanisms of response have been replaced by emotional processes which involve the appraisal of environmental events -including here the 'milieu interieur'-in terms of their significance for the individual. The efferent part of emotions involves a physiological response to set the organism into the adequate state to deal with the environmental circumstance, and an action tendency or motor attitude (and in some extreme cases a whole pattern of action). Emotions usually include expressive behaviour accomplishing primarily the social function of communicating the emotion to other members of the species. The subject eventually becomes aware of the emotional response, and this generates the experiential or subjective aspects of emotion.

1.4.2. The emotional cycle

A fundamental idea in the understanding of the emotional processes (and in general all psychological phenomena) is that emotions are interactive, dynamic processes that occur over time. Neisser (1976) has proposed a model of perception, "the perceptual cycle", in which these notions of interaction between individual and environment and dynamism are central. According to Neisser (1976), the organism possesses "anticipatory schemata that prepare the perceiver to accept certain kinds of information rather than other" (p. 20), and guide the

constructive process that perception is. Neisser (1976) conceives perception as a cyclical sequence of exploratory movements, information pick-up, exploratory action again, more information pick-up, and so forth. As Neisser has pointed out the interactive process is more obvious in the haptic perception than in vision or audition, although the perceptual cycle in the different sense modalities is essentially identical. Arbib (1981) has used Neisser's perceptual cycle model ('action-perception cycle' is called by Arbib) to analyze visuomotor coordination in biological organisms and artificial intelligence systems. Arbib (1981) emphasizes that perception cannot be understood "unless it is embedded within the organism's ongoing interaction with its environment" (p. 1459). Perception does not involve only the decodification of the physical features of the objects, but it also includes "the activation of routines for the interaction with the object (motor schemas)", although it "does not necessarily involve execution" (Arbib ,1981, p. 1459). Arbib (1981) has extended Neisser's model by conceiving the exploratory movements in a more general fashion as action upon the environment as well as action to guide further information pick-up. Neisser-Arbib framework illustrates well some fundamental features of how an organism (living organism or artificial intelligence system) interacts with a complex environment. Emotion is a special type of organism-environment interaction and as such consists in a cyclical process (the emotional cycle) of bidirectional interactions.

In what follows I will attempt to analyze briefly the elements of the 'emotional cycle'. In this analysis the information-processing paradigm has been adopted. Information-processing (or cognitive) models characterize organisms as information-processing systems, and the functioning of these systems is described in terms of a series of

processing mechanisms or processing stages. The concern of the information processing paradigm is "the flow of information/knowledge within the organism and between it and its environment" (Mandler, 1985, p.19). Although cognitive psychology has now been for some time the mainstream psychology, it has neglected until recently the study of emotion (Norman, 1980). A decade after Norman (1980) identified emotion as one of the challenges of cognitive psychology the situation has changed substantially, and today it is possible to say that cognitive psychology of emotion is well established, and its contribution is now starting to be apparent in applied and clinical areas (see Williams et al, 1988).

Fisher et al (1990) states that "contemporary emotion theorists and researchers agree substantially on the broad nature of emotion" (p. 83). This is basically true, and the conception of emotion presented here is essentially within the boundaries of this consensus.

The first step in the processing of emotional stimuli is carried out without the participation of consciousness. There is extensive evidence that unattended stimuli are processed, and that this nonconscious processing includes high-order structural and semantic analysis; compelling evidence also exists demonstrating that emotional responses can be evoked without the mediation of consciousness. This evidence has been reviewed by various authors: Zajonc (1980), Dixon (1981), Froufe (1985), Williams et al (1988) amongst others. There is a continuous nonconscious emotional processing of stimuli. This emotional processing implies the perception (which necessarily involves cognitive appraisal) of sensory stimulation in terms of its 'emotional significance'. That is, in terms of the consequences for the survival and

the welfare of the individual or/and the species, the requirements imposed on the bodily functioning to keep the homeostatic balance, and the needs of autonomic response and motor readiness to set the background that enables the individual to produce an adequate response to cope with the situation. The nonconscious processing of certain stimulus may result in an emotional response, which will consist of physiological activation and a motor attitude or response, and will possibly be accompanied by a call on central controlled processing resources. Ohman (1986), for example, has shown that subconscious presentation of conditioned stimuli elicits the physiological response of fear. Mathews and McLeod (1986) have shown that nonconscious processing of unattended material (a dichotic listening procedure was used) captured processing resources and interfered with the conscious task when the unattended stimulus had anxiety-producing value for the subjects.

It is also conceivable that the emotional reaction sets the organism in a new tonic emotional state (which we will call 'mood') without interfering with on-going conscious processing. The new affective state or mood will influence subsequent processing of emotional material. There is strong evidence that mood influences cognition and biases the processing of emotional material (e.g. Bower, 1981, Forgas and Bower, 1987).

The first stage of nonconscious emotional processing may be followed by subsequent steps characterized by a 'more conscious controlled processing'. The emotional perception of a stimulus may call upon the central channel interfering with the task being presently carried out consciously. In some cases, the emotional reaction will trigger a 'raising

of consciousness', in which the individual will become aware of the emotional stimulus at the same time as he feels the physiological reaction. A secondary more conscious and controlled 'appraisal' of the situation will take place (Frijda, 1986) . Conscious appraisal of the stimulus may change the valence assigned to it by the first automatic analysis. Conscious processing may result in the deployment of conscious controlled strategies to cope with the situation. These procedures or strategies may eventually become automatic (see Shiffrin and Schneider, 1977). The individual may engage in voluntary controlled behaviours which are incompatible with, limit, or even oppose the motor response elicited by the emotional stimuli. The subject may purposely and voluntarily flee from the stimuli, 'short-circuiting' the emotional stimulation. The feedback loop by which the organism monitors itself and which effectively determines emotional output may be interfered. Autonomic response and expressive behaviour are used to monitor the emotional state of the organism. Prevention or disruption of feedback from expressive output has been shown to alter the intensity of emotional feelings (e.g. Strack et al, 1988) and the physiological response to incoming emotional stimuli (e.g. Lanzetta et al, 1976).

The cyclical sequence of appraisal, physiological reactions and actions upon environment give origin to the subjective experience of emotion. The experience of emotion is the construction in consciousness of the concatenation of the different elements of the emotional process (appraisal, physiological response, etc.). As Mandler (1985) has explained, this conscious construction is always a holistic unitary experience. Consciousness has limited access to the psychological processes. Therefore, verbal reports on own emotions will consist to a

variable extent in plausible rationalizations (Nisbett and Wilson, 1977, Froufe, 1985).

This process-type account of emotion can be complemented with the description of the different levels of the emotional response that the emotional cycle builds up. Fisher et al (1990) have proposed a hierarchical organization of the emotional states. At the top of the hierarchy, there are two basic responses (negative or positive) of the organism to emotional stimulation. This layer of the hierarchy coincides with Arnold's conception of emotion (Arnold, 1960) as "the felt tendency toward anything intuitively appraised as good (beneficial), or away from anything intuitively appraised as bad (harmful)" (p. 182). For Arnold (1960) this attraction or aversion is accompanied by a pattern of physiological changes organized towards approach or withdrawal. The pattern differs for different emotions.

In the second layer of the hierarchy Fisher et al (1990) locate a series of 'basic emotions'. They are considered basic because they have been found in most cultures. These emotions are for Fisher et al (1990): anger, sadness, fear -in the family of negative emotions-, love and joy - in the family of positive emotions.

In the last layer there are more complex socially constructed emotions, derived form the basic emotions. This third group of emotional states will differ in different cultures and social groups.

Without adhering totally to the Fisher et al. (1990) model, which is more comprehensive and specific, I take the idea of the hierarchical organization of emotional categories, and that of an increasing influence

of social learning (via, for example, learned expectancies) as we advance through the levels of the hierarchy. This model marries two schools of thinking: that which since Darwin has considered emotions as biologically determined response patterns, and that of those who have emphasized the social construction of emotion. More important for our purposes, the 'products' of the different stages of the emotional cycle fit well in this hierarchical organization. The primary appraisal of the emotio nal stimulus will result in a first or second layer response, depending on the complexity and specificity of the stimulus. Secondary appraisal and concious regulatory activity will result in a third layer response.

1.4.3. Psychology of emotion and alcohol research

Not long ago, Chick (1984) wrote that "if we are to advance our understanding of intoxication, we will need better theories of the relation between perception, thought, mood and behaviour. [...] In these times we cannot merely say that alcohol 'disrupts high processes' and 'leaves the instincts free to roam'" (p. 60). In general, alcohol research has neglected the theoretical side. For example, to find the first serious attempt to define the concept of expectancy in this context we have to wait, after many years of empirical research into the association between expectancies and drinking, until 1987 (Goldman et al, 1987). The demonstration of the role of the psychological factors in the determination of behaviour after drinking led to seek theoretical explanations of the phenomenon. The proposed explanation was based on Schachter's attributional theory (Schachter, 1964). Schachter highlighted the importance of the cognitive appraisal and the causal attribution of the physiological response elicited by the emotional

stimulus in the determination of the final emotional response. Schachter focused on the attributional mechanism in order to explain the results of his well-known experiment, but if we read Schachter carefully we realize that he was aware of other aspects and stages in the emotional process. Thus, we can read: "The perception cognition 'figure with a gun' in some fashion initiates a state of physiological arousal; this state of arousal is labelled 'fear'" (Schachter, 1964, p. 51). In terms of the model presented here we can 'translate': The stimulus 'figure with a gun' (even if presented subliminally) elicits an emotional reaction including a physiological response, also probably (although Schachter does not mention it) a disposition to flee (i.e. motor attitude) and a call upon central resources (attention is attracted towards the stimulus); a conscious secondary appraisal will label the reaction as 'fear'. Additionally, the person facing the situation might also have expectancies about the probable behaviour of the individual holding the gun, about what other people or society in general expect from him in that situation, and all this, not only the attachment of the label 'fear', will influence his appraisal and determine his behaviour, and generate a more complex socially determined emotion. In sum, Schachter's model can be easily integrated in the more comprehensive framework presented here.

Recently, a number of partial theories -Wilson (1988) has referred to them as 'mini-theories'- of the effects of alcohol have appeared (see the recent volume compiled by Blane and Leonard, 1987). These theories highlight different aspects of the effects of alcohol (e.g. 'stress dampening theory', 'self-awareness theory', etc.). The data upon which they are based should be integrated in a more comprehensive model.

The framework of emotion presented here turns generic questions like 'does alcohol disinhibit?' or 'does alcohol have an anxiolytic effect?' into more specific objectives of research. For example, using the prior hypothetical situation, we could ask: does alcohol influence the primary reaction to the emotional stimulus 'figure with a gun'?, does alcohol influence the conscious evaluation of the risks involved?, are there specific expectancies concerning the situation 'facing a man with a gun when I have been drinking' that might influence the appraisal of the situation?, etc.

This thesis represents an attempt to investigate several aspects of the effects of alcohol intoxication on emotional behaviour. I shall focus on two aspects of emotion which have particularly attracted the interest of the researchers of the effects of alcohol: sexual response and anxiety (or stress) response. The experimental literature on these two areas will be reviewed and several experiments will be carried out in the light of the framework of emotion exposed above.

Chapter 2

ACUTE EFFECTS OF ALCOHOL ON SEXUAL RESPONSE

2.1. Introduction

Wilson (1981) began a review in this area of research by pointing to the scarcity of scientific studies on the relationship between alcohol and sexual behaviour. He observed that, "in the absence of objective information, literary insights have passed for authority in both public and professional spheres" (p. 3). The disinhibition theory, applied in this case to sexual behaviour , has remained the conventional view on this issue. The idea that alcohol disinhibits sexual responding has existed at least since the days of classical Rome and the first Popes (see MacAndrew and Edgerton, 1969). The disinhibition theory states that alcohol releases sexual impulses which are otherwise kept under the control of inhibitory forces. Depending on the theoretical persuasion of the author, the inhibitory agents have been seen as "higher brain functions" (e.g. Jackson, 1958) or as psychodynamic mechanisms (e.g. Cohen, 1967).

Experimental research on the acute effects of drinking on sexual response has been carried out mainly over the last fifteen years. In what follows I will attempt to review this research. Two sources of evidence can be distinguished: on the one hand, a number of studies have investigated retrospective reports of the acute effects of alcohol on sexual behaviour; on the other hand, since the mid 1970s a series of experimental studies have been carried out which have investigated the acute effect of ethanol on objective measures of sexual response.

2.2. Survey research: people's views on the effects of alcohol on sexual behaviour

Wilson (1981) has reviewed relevant data from questionnaire surveys and concluded that these studies "do suggest that alcohol increases sexual satisfaction and is related to greater sexual activity in both men and women" (p. 6). It could be argued, however, that this conclusion does not reflect the whole story. It is true that survey research has revealed that a high proportion of people think that alcohol exerts positive effects on their sexual behaviour. For instance, 45% of the men and 68% of the women that answered the questionnaire on sex published in Psychology Today agreed that alcohol "greatly or somewhat" enhanced their enjoyment of sex (Athanasiou et al, 1970). However, although this study provides what are perhaps the figures that most strongly support Wilson's conclusion, it must be noted that 42% of the men and 21% of the women in this survey indicated that alcohol "greatly or somewhat" decreased sexual enjoyment. Therefore, it could be claimed that the only possible conclusion to be drawn from these studies is that a significant proportion of the population believe in the aphrodisiac effects of alcohol, though there is still a large number who do not hold that opinion. This type of research also shows that views on this matter are highly variable, the percentage of those who report aphrodisiac effects of alcohol being substantially different across studies (see Wilson, 1981). These discrepancies may be related to the characteristics of the populations studied and also to the different questions asked. Evidence from a recent study (Klassen and Wilsnack, 1986) in which 917 American women were surveyed concerning 'sexual experience and drinking' illustrates this point well. Sixty per cent agreed that after drinking they "felt less inhibited about

sex"; and 45% declared that sexual activity "was more pleasurable" when they had drunk. However, only 22% agreed that they "became sexually forward" and an even smaller proportion, 8%, said that they became "less particular" in their choice of partner. It seems clear that the term sexual arousal is not an unitary concept but it has a variety of referents and that alcohol can influence the diverse aspects of sexual responding differently.

Brown et al (1980) developed the "Alcohol Expectancy Questionnaire" using factor analysis. Enhancement of sexual performance and experience is one of the dimensions of people's expectancies about alcohol effects they found. Interestingly, they also found that heavy drinkers held stronger expectancies in this sense than moderate consumers.

In sum, the view of alcohol as an aphrodisiac seems to be widely held, although far from universal; the role of different factors - actual experience, folk wisdom, etc.- in making people hold such a belief remains to be understood. Further research should clearly distinguish between what individuals think are the normal effects of alcohol on people's sexual behaviour and what they believe are the consequences of drinking on their own behaviour. Rohsenow (1983) has shown that people tend to expect alcohol to affect others more than themselves, both for positive and negative effects. This pattern of expectancies appeared to be particularly true for light drinkers, while moderate and heavy drinkers tended to expect as many positive effects from alcohol as they thought other people experienced. It is also important to distinguish between the effects of alcohol on the various aspects or components of sexual behaviour, e.g. feelings of arousal,

enjoyment of sexual activity, performance, etc.

Questionnaire research is valuable on its own. It will always be interesting to know people's views on this issue, for several reasons, not the least important the possibility that beliefs, no matter how poorly supported by reality, may actually influence behaviour. However, retrospective self-reports are not a good substitute for evidence gained through direct experimentation. In a classic paper, Nisbett and Wilson (1977) demonstrated that consciousness does not have access to all psychological processes, and that people tend to construct plausible explanations for the causes of their own behaviour, 'telling more than what they really know'.

2.3. Experimental study of the acute effects of alcohol on human sexual response

2.3.1. The objective measurement of sexual arousal

The experimental study of human sexual response has been boosted by the development of techniques that allow objective measurement of sexual arousal. Genital measures have been shown to be the only psychophysiological indices that are specific to sexual arousal (Zuckerman, 1971). Penile diameter can be measured unobtrusively by means of a strain gauge consisting of a fine tube of silicone filled with mercury. Changes in penile diameter are reflected in variations in the electrical resistance of the mercury (Bancroft, 1974). Vaginal vasocongestion, measured through a photoplethysmograph, has been the most frequently used psychophysiological index of sexual arousal in women. Objective research of sexual response in women has lagged

behind that in men. Measurement of female sexual response has been shown to be more complex than that of men, and, consequently, research in women has rendered less clear-cut results (Hoon, 1984). This may be one of the reasons why very few studies have looked at the effects of alcohol on female sexual arousal, although a sexist bias which has neglected the study of female sexuality may have contributed to this scarcity.

The relatively small number of studies in this area makes it possible to examine each published study. The focus will be on studies using objective measures of sexual arousal, although some that did not use them will also be included, on the grounds that they provide evidence to clarify points raised in the more psychophysiologically-orientated work. As the experimental work in this thesis will use male subjects, the main concern of this review will be the literature dealing with alcohol and male sexual response. However, the few studies that have been carried out in women will also be mentioned, for the sake of completeness.

2.3.2. The effects of alcohol on male sexual response

Three studies published in 1976 examined experimentally the acute effects of alcohol on sexual response in normal male volunteers. Farkas and Rosen (1976) examined the penile response to heterosexual erotic videos in 16 male student volunteers at three different blood alcohol concentrations (BAC), i.e. 25, 50 and 75 mg/% (actual doses administered are not reported), using a repeated measures design, where each subject saw the same film at each level of alcohol in blood. Low BAC had a facilitative effect on maximum tumescence and

tumescence rate (maximum diameter change divided by latency to reach that point), although these effects did not reach statistical significance. BACs of 50 and 75 mg/% exerted an inhibitory action over erectile response, the effect being greater at 75 mg/%. A significant linear trend accounted for 87% of the variance of the effect of alcohol on erectile response.

Rubin and Henson (1976) examined the effects of three different doses of ethanol in penile tumescence on three different conditions: (1) while watching an erotic video, (2) trying to inhibit sexual arousal while seeing an erotic video, and (3) trying to get sexually aroused in the absence of external stimuli. The complex design involved each subject undergoing three experimental sessions with an interval of seven days between each. In the first and third sessions subjects received a placebo beverage. In the second session, eleven subjects received alcoholic drinks, and the other five were given placebos. During the experimental sessions subjects watched four different films, each twice, first under instructions to relax and enjoy it and then with instructions to inhibit sexual arousal. After the second presentation of each film, alcohol group subjects were administered a drink containing 0.5 or 0.6 g/kg of alcohol, which was consumed in three minutes. Thus, each subject received three of these doses. After the fourth film, the self-arousing task took place for ten minutes. Hence, subjects were tested after the consumption of three different amounts of alcohol: 0.5 or 0.6, 1.0 or 1.2 and 1.5 or 1.8 g/kg. The self-arousing task was tested only under the highest dose, though. The small and moderate doses had little or no effect on penile response, whereas the high dose produced a significant depressive effect on erection. No significant effect was found on subjects' ability to inhibit sexual arousal. The high dose of

alcohol also produced a very large decrease in sexual arousal when subjects were instructed to become sexually aroused in the absence of overt erotic stimuli. As insufficient time was allowed for each dose to be absorbed, it is difficult to draw any conclusions about the dose-effect relation from this study. It is hard to say whether the significant effect found for the high dose was due to the action of this amount of alcohol or due to the previous dose which had only just reached the blood stream. Despite the clear reducing effect of alcohol most subjects believed that alcohol had enhanced their responses.

In the study by Bridell and Wilson (1976) 48 male undergraduates were allocated to one of four different dose-groups: .08 g/kg (placebo), .4 g/kg, .8 g/kg or 1.2 g/kg. Half of each group were told that the amount of alcohol they had consumed "usually reduces sexual arousal" in response to the erotic video they were about to be shown. The other half were led to expect a positive effect. This expectancy manipulation did not significantly affect penile tumescence. A significant negative effect of alcohol dose on erectile response was found.

The inhibitory effect of alcohol has also been found in alcoholics. Eight chronic alcoholic men were the subjects in the study of Wilson et al (1978). Four doses of alcohol were administered in four different experimental sessions. In counterbalanced order subjects consumed 0.08 g/kg (placebo), 0.4 g/kg, 0.8 g/kg and 1.2 g/kg, prior to watching two explicit erotic, heterosexual and homosexual films. The two high doses considerably reduced the erectile response. A significant negative linear effect of increasing alcohol doses on both films was found. The 0.4 g/kg dose increased maximum penile tumescence, particularly

during the homosexual film, although this effect did not reach statistical significance.

These four studies demonstrated that alcohol has an impairing effect on penile tumescence, and that this effect seems to be dose-related. These experiments were followed by a series of studies examining also the influence of the belief of having drunk alcohol using the Balanced Placebo Design (BPD).

Wilson and Lawson (1976) used a BPD to study the effects of alcohol and the belief of having consumed alcohol on the erectile response to two different erotic films, one showing heterosexual interactions, the other a male homosexual interaction. Forty male undergraduates participated in the experiment. No effect of the dose of alcohol administered (0.5 g/kg) was found. Subjects induced to believed that they were drinking alcohol, irrespective of the actual content of the drink, showed significantly greater maximum percentage increase in penile diameter on both films.

Bridell et al (1978) conducted a BPD study with 48 undergraduate male social drinkers, using an alcohol dose of 0.5 g/kg. They measured the erectile response to three different taped narratives describing mutually enjoyable intercourse, forcible rape and nonsexual sadistic aggression. Additionally, subjects were asked to attempt to increase their level of sexual arousal by any means other than physical manipulation of the genitals and were "encouraged to employ vivid sexual fantasy" for that purpose . Maximum changes in penile diameter from pre-tape baseline were calculated for each type of narrative and for the self-arousal period. When the results for the

three narratives were analyzed as a whole, a highly significant positive effect of the belief manipulation was found. Although the main effect of alcohol was not significant, the interaction effect was, suggesting that "subjects who received alcohol were somewhat more influenced by the instructional set than the subjects receiving the placebo" (p. 423). When responses to the various tapes were analyzed independently, it was found that belief manipulation significantly affected penile tumescence in response to deviant narratives but not to the heterosexual intercourse tape. No effects of alcohol or interaction were found when the tapes were considered separately. Self-induced arousal was not affected by alcohol or instructional set.

Lansky and Wilson (1981) examined the effects of alcohol and expectancy on sexual response and, additionally, explored the cognitive processes that may mediate these effects. Twenty minutes after the administration of the drinks (containing 0.6 g/kg for half of the people) subjects were presented with 30 slides - 10 heterosexual, 10 homosexual and 10 nonsexual - and were asked to evaluate each one on several dimensions: arousal, obscenity, pleasure, eroticism and interest. Later, they listened to heterosexual, homosexual and nonsexual narratives over headphones. While they were listening to the stories, a number of brief tones appeared at a variable interval 30 sec schedule. Subjects were instructed to press a button whenever they heard the tone. Reaction time was measured as an index of attention. Penile tumescence was recorded continuously during the narratives. Recognition memory for the pictures and narratives was also assessed. Erectile response to narratives was significantly affected by the belief of having drunk alcohol only for those who were high in sex guilt. Subjects spent significantly more time viewing the heterosexual slides

than the homosexual ones and in turn the time spent looking at these was longer than the time spent on the nonsexual slides. Reaction time to the tones was significantly shorter during the nonsexual narratives. Recognition memory for the heterosexual slides was significantly better than memory for the nonsexual slides, which in turn was more accurate than for the homosexual slides. Drink content affected recognition memory for visual material significantly: those who received alcohol performed worse. Recognition memory for narratives was significantly affected by the expectancy manipulation. Those who were told that they were drinking alcohol showed more accurate recall. Those who actually received alcohol performed worse than those who had placebo, although this effect only approached statistical significance ($p< 0.07$). No significant correlation between penile tumescence, reaction time and recognition memory was found.

In a study in which objective measures of erection were not taken, Lang et al. (1980) investigated the effects of a dose of alcohol of 0.36 g/kg and the belief of having consumed an alcoholic drink -using BPD - on time spent viewing slides of diverse erotic content. The experiment examined the moderating effect of individual differences in sex guilt. After drinking, subjects were asked to evaluate a number of erotic photographic slides. Subjects who were told that their drinks contained alcohol reported significantly higher subjective sexual arousal. Viewing times increased as a positive linear function of pornographic ratings of the slides except for those who thought they were drinking a soft drink and scored high on the sex guilt questionnaire.

The disinhibitory effect of the belief that one is under the influence of alcohol found in several studies has been explained on the grounds of

the self-exoneration that people can achieve by attributing the responsibility for their sexual behaviour to the action of alcohol (see below). McCarty et al (1982) speculated that the pharmacological action of alcohol might make a greater contribution to sexual arousal if "conventional" sexual stimuli were used. They argued that, although the exonerating effects of drinking may work when deviant, highly obscene stimuli are used or when the subjects are high sex-guilt individuals, the preponderant influence of the instructions about the content of the drink may not be found when subjects do not have any reason to consider their responses to be unacceptable. In an experiment where a BPD was used, they found that subjects (of both sexes) who had drunk alcohol (0.35 ml/kg), but had been told that their drinks contained only tonic, were the ones who rated a number of "conventional" erotic slides taken from Playboy, Penthouse, Playgirl and fashion magazines as most sexually arousing. McCarty et al. (1982) interpreted this finding in the light of Zillman's theory of "transfer of excitation". According to Zillman (1978), physiological changes caused by particular stimuli can be wrongly attributed to different stimuli resulting in an enhanced perception of the arousal response to this second source. Thus, McCarty et al's (1982) interpretation of their results is that "when subjects were unaware of the alcohol intoxication, the alcohol excitation transferred to the evaluation of the photographic slides" (p. 983).

Abrams and Wilson (1983) investigated the effects of alcohol and expectancy on self-regulation of sexual behaviour using the delay-of-gratification paradigm. The BPD was utilized and a dose of 0.45 g/kg of ethanol administered. Additionally, there was a fifth experimental group who received 0.9 g/kg of alcohol and was told that they would

be given an alcoholic drink. Subjects were shown a brief (45 seconds) priming erotic film which was followed by the "optional waiting period". The subject was told that he was going to be presented with another erotic video and that the duration of that film would be proportional to how long they were able to delay the start of it (delay-of-gratification). The results showed that subjects who believed they had drunk an alcoholic beverage irrespective of the real content of the drinks waited longer to see the erotic film. These subjects reported less embarrassment, more pleasant feelings, and more sexual thoughts and feelings during their delay intervals. Neither dose of alcohol significantly affected these variables. In Abrams and Wilson's view the conflict subjects faced was between an inhibited response of shortening the delay period to see a smaller amount of film and the more hedonistic, disinhibited option of a longer delay. Thus, Abrams and Wilson (1983) interpreted the results as indicating that the believed intoxication reduced self-control and made subjects "give in" to see greater amounts of the film. Neither alcohol nor belief affected penile tumescence in any phase of the experiment. No effect of the Mosher Sex Guilt Inventory scores was found.

Barbaree et al (1983) conducted a study using the BPD, in which 32 male social drinkers (graduate students) were presented with descriptions of mutually-consenting sexual intercourse and rape. They used a dose of 0.63 ml/kg. No effect of instructional set or alcohol on erectile response was found. Expectancy manipulation was not successful for the group who were told that they were receiving a soft drink and who actually received an alcoholic beverage, which makes results difficult to interpret. After controlling statistically for the differences in the amount of alcohol they thought they had ingested, it

was found that subjects who had the nonalcoholic drink tended to show greater differences between their penile response to mutually-consenting intercourse (always larger) and rape materials than subjects who drank the beverage containing alcohol.

Wilson et al (1985) set up an experiment to investigate the effects of expected and actual alcohol drinking on sexual response to erotic narratives, aiming also to ascertain the cognitive processes that may mediate these effects. Thirty two male undergraduates were randomly allocated to the cells of a BPD; those in the alcohol groups consumed 0.6 g/kg of alcohol. A dichotic listening task was used. Subjects were presented with erotic and non-erotic stories through the non-attended channel, while they carried out numerical tasks of two levels of difficulty, thought to demand two different degrees of attention, which were presented in the attended channel. Attentional demands significantly affected percentage increase of penile diameter during the erotic narratives. Subjects were significantly more aroused under the low attention condition. A significant attention demand by drink content interaction effect revealed that the high-attention demanding task reduced erectile response only for those subjects who actually received alcohol. Instructional set also interacted with the attention demand factor. Under low-attention demand those who believed that they had drunk alcohol showed greater arousal than those who believed they were sober. The degree of attention required by the arithmetic tasks did not affect penile tumescence in response to erotic narratives when subjects had not consumed alcohol, which is at variance with the finding of the influential study by Geer and Fuhr (1976), who, using a similar dichotic listening paradigm, found a suppressant effect of attention demands on erectile response.

Twenty two subjects who had already taken part in the Wilson et al. (1985) experiment participated in a second study also involving a BPD (Wilson and Niaura, 1984). Subjects listened to an erotic narrative and penile tumescence was measured. They had been previously instructed to try "as hard as they could to suppress sexual arousal" during the presentation of the erotic description. No effect of instructional set about drink content was found. Alcohol actually administered (0.6 g/kg) significantly reduced the latency to onset of erectile response and to peak level of tumescence.

George and Marlatt (1986) found that subjects who believed that they had drunk alcohol spent more time looking at violent, erotic and violent-erotic slides in a procedure of ad lib viewing. These subjects rated more highly the erotic and violent-erotic slides. There was no effect of alcohol (0.4 g/kg).

The effects of alcohol on ejaculation have also been studied. Malatesta et al (1979) administered four different doses (resulting in BAC of 0, 30, 60 and 90 mg/ 100 ml) in a repeated measures design. Subjects were asked to masturbate while viewing explicit erotic videotapes. Results showed that the higher the BAC the longer it took to ejaculate; a linear trend explained 95 % of the variance. Additionally, subjects reported decreased subjective arousal, reduced pleasure and lessened intensity of orgasm at higher levels of BAC. Ten of 24 subjects failed to achieve orgasm at the BAC of 90%.

2.3.3. The effects of alcohol on female sexual response

In order to give a complete account of this area of research the few studies conducted on women will be mentioned briefly.

Wilson and Lawson (1976) used a repeated measures design to investigate the effects of four different doses of alcohol (0.05, 0.25, 0.50 and 0.75 g/kg) on sexual response to an erotic film. Half of the subjects were told that a well-known effect of alcohol was to increase the sexual response; the other half was told that it was expected that alcohol would decrease their sexual responses to the erotic film. A significant negative linear effect of the amount of alcohol consumed on plethysmographic measures of vaginal pressure pulse was found. No main effect of instructional set appeared. There was no significant correlation between subjective arousal and measures of vaginal response. Alcohol consumption did not influence the subjective ratings of sexual arousal.

A BPD was used in a second experiment by Wilson and Lawson (1978) in which women were presented with two kinds of erotic films, heterosexual and homosexual in content. There was no significant effect of instructional set. Those who had consumed alcohol (0.4 g/kg), irrespective of what they were told, showed significantly less increase in vaginal pressure pulse during the heterosexual film. Subjective rating of intoxication were significantly correlated with self-reported sexual arousal during both films.

Using a repeated measures design Malatesta et al (1982) administered

four different doses of alcohol (0, 25, 50 and 75% BAC) to 18 female university students. Subjects watched explicit sexual films and were asked to masturbate to orgasm. Higher BACs were associated with longer orgasmic latencies and decreased intensity of orgasmic contractions. However, at the moderate and the high doses subjects reported significantly greater sexual arousal and orgasmic pleasurability.

2.4. Discussion

Alcohol per se seems to have an impairing effect on the physiological sexual response. This has been shown both when subjects were presented explicit stimuli (Farkas and Rosen, 1976, Bridell and Wilson, 1976, Wilson et al, 1978) and when arousal was self-induced through fantasy (Rubin and Jenson, 1976). The reducing effect of alcohol appears to be dose related: the higher the dose the more pronounced the depressive effect is. Farkas and Rosen (1976) found that a BAC of 50 mg/% significantly reduced penile tumescence in response to erotic stimuli; a dose of 0.8 g/kg has been found to be sufficient to produce a significant negative effect on genital response in males (Bridell and Wilson, 1976, Wilson et al, 1978). Doses as low as 0.4 g/kg have been shown to exert a significant depressive influence on vaginal vasocongestion. Below these levels of intoxication, alcohol may even have a slight positive effect: a small positive effect of ethanol has been found at 25 mg/% BAC (Farkas and Rosen ,1976) and after drinking a dose of 0.4 g/kg, although in neither case did these effects reach statistical significance.

The belief of having drunk an alcoholic drink tends to produce greater increases in penile tumescence in response to erotic stimuli. The same

has not been found in women, but it has to be noted that only one study testing this has been carried out. The nonpharmacological - placebo- effect of drinking can happen independently of the actual alcohol consumed (e.g. Wilson and Lawson, 1976). However, an interaction between this belief-generated effect and actual intoxication has also been found: in Bridell et al. (1978) subjects who had consumed alcohol were more influenced by the instructional set.

The effect of the belief of having drunk alcohol has been found to be more pronounced in subjects who scored higher in sex guilt (Lang et al, 1980), and also when erotic stimuli were deviant (Bridell et al 1978; George and Marlatt, 1986). The belief that one has consumed alcohol tends to produce a positive effect in both physiological and subjective sexual arousal. However, alcohol can influence physiological and subjective arousal in opposite directions: subjects may persist in their belief that alcohol enhances sexual response even when the alcohol had few minutes earlier an objective reducing effect on their responses (Runbin and Henson, 1976). A positive effect of alcohol has been found when subjects were asked to suppress sexual arousal (Wilson and Niaura, 1984).

Hull and Bond (1986) carried out a meta-analysis on nine BPD studies looking at sexual arousal (they did not include Barbaree et al. (1983) study, published in an European journal) and concluded that the belief of having drunk alcohol had "a sizeable statistically significant effect of increasing sexual arousal" (p. 353) both on self-report and on physiological measures. On the other hand, the results of the meta-analysis evinced a small nonsignificant effect of alcohol. It is important to observe, however, that the direction of the alcohol effect is not

consistent across studies: alcohol increased sexual arousal in four studies and had a negative effect in five. Various factors could explain this inconsistency. First of all, it should be noted that the dose levels used in these studies usually ranged between 0.4 g/kg (which has been found to produce a small nonsignificant positive effect on penile tumescence) and 0.8 g/kg (which has been shown to have a consistent reducing effect). Thus, it could be argued that the doses employed in these studies lie in an intermediate region in which it is difficult to find consistent effects. The different experimental situations and variables manipulated, besides alcohol and belief, could also explain some of the inconsistencies. One may legitimately question to what extent this inconsistency reflects theoretically revealing variability in the effects of ethanol. On the other hand, one may also question the validity of a crude meta-analytic statistic estimating the effect size, based on nine studies, when other different variables in addition to that under scrutiny in the analysis have been used in several of the studies, and when different doses have been employed.

An explanation of the effects of the belief that alcohol was consumed has been formulated within the framework of "Social Learning Theory". Two mechanisms have been proposed within this approach (Wilson, 1977,1981). Firstly, it has been argued that believing that one is intoxicated may enhance sexual response because it gives the individual the opportunity to attribute responsibility for "inappropriate" sexual arousal to alcohol. Secondly, it is thought that people may learn that in those circumstances where alcohol is involved behaviour which is otherwise socially unacceptable is tolerated.

These two mechanisms could account for the disinhibition of social,

voluntary controlled, sexual behaviours: behaviours that, depending on the circumstances and context, could range from a sexually-orientated social approach to indulgence in "unusual" sexual practices. This model, however, fails to give a satisfactory explanation for the physiological sexual arousal, which is more resistant to voluntary control. The belief that alcohol has been drunk may influence the processing of sexual stimuli. The identification of possible mediating cognitive processes was the objective of Lansky and Wilson (1981). More research is needed in this direction.

BPD studies have been useful to make us aware of the importance of purely psychological processes in determining the consequences of drinking. "Social Learning Theory" explanation of the placebo effect of alcohol neglects any possible pharmacological action of the ethanol. The known pharmacological action of alcohol, i.e. the effect of reducing sexual response, goes against the psychologically generated aphrodisiac consequences of drinking. What is then the origin of the extended belief that alcohol is aphrodisiac? The 'Transfer of Excitation' model predicts a pharmacological aphrodisiac effect of alcohol. On the other hand, Wilson and Niaura (1984) found a situation in which alcohol had a pharmacologically-mediated arousing effect. This finding will be closely examined in the next chapter.

One of the main problems facing research in this area is conceptual. Sexual arousal is a vague term referring to a wide range of such diverse phenomena as physiological arousal, subjective states, social behaviour, etc. Sexual arousal must be understood as an emotion comprising the typical elements of emotional phenomena interacting in a complex way (see Chapter 1). Several recent papers have pointed out

the complex nature of sexual arousal and indicated the different components that should be distinguished and the various ways in which they interact (e.g. Walen, 1980, Dekker et al, 1985). The effects of alcohol on sexual arousal can only be conceived as the result of the action of ethanol on the different components it consists of and their interaction.

Chapter 3.

AN EXPERIMENT EXAMINING THE DISINHIBITORY EFFECTS OF ALCOHOL ON SEXUAL RESPONSE

3.1. Introduction

It was concluded in Chapter 2 that the belief that alcohol enhances sexual response is widely, although not universally, held. However, experimental research has demonstrated that alcohol per se inhibits sexual response. To solve this paradox it has been suggested that the aphrodisiac effects of drinking stem from psychological factors, e.g. socially learned expectancies. In chapter 1, it was put forward the idea that, although psychological factors are without doubt very important, if alcohol has kept its reputation as a disinhibitory agent for so long and in so many cultures, that may be because of its real pharmacological effects. It is possible that alcohol has an aphrodisiac effect only when drunk in certain circumstances, or in interaction with certain factors. The study by Wilson and Niaura (1984) suggests a situation in which alcohol might have a significant disinhibitory effect. In this experiment, in which subjects were asked to suppress sexual arousal while listening to an erotic narrative, alcohol reduced the latency to onset of erectile response and to maximum level of tumescence, indicating a pharmacologically-mediated aphrodisiac effect of alcohol.

The purpose of the present study was to replicate and investigate further Wilson and Niaura (1984) findings. If drinking precludes voluntary inhibition of sexual arousal, as Wilson and Niaura found, it would have not only academic interest but also forensic implications.

Wilson and Niaura (1984) seemed aware of the possible repercussions of their finding and explicitly called for an independent replication.

Different erotic stimuli will be used in the present study. Wilson and Niaura (1984) made their subjects listen to taped narratives; filmed erotic material will be used in this study. The equivalence of different types of sexual stimulation should not be assumed, as there is evidence that different psychophysiological mechanisms may be involved in the response evoked by different types of sexual stimulation. It has been found, for example, that response to video-taped visual erotic material is androgen-dependent whereas arousal self-induced through fantasy is not (Bancroft and Wu, 1983).

The main aim of the present experiment was to examine the effects of alcohol on the voluntary control (inhibition and generation) of sexual response. Accordingly, a second task was included. Subjects were asked to become sexually aroused through imagery in the absence of external stimuli. A general disinhibitory effect of alcohol would result in an increased sexual response in both tasks. Any specific impairing effect of ethanol on the ability to control (inhibit) sexual reaction would yield an increment of sexual arousal on the inhibitory task, while as previously reported in the literature (Rubin and Henson, 1976) alcohol would reduce sexual responding on the self-arousing task.

Since the primary objective was the replication of Wilson and Niaura's findings, inhibitory and self-arousing tasks were not counterbalanced, in order to make results concerning the former directly comparable.

Wilson and Niaura (1984) did not find any effect of the induced belief

of having drunk alcohol. However, it must be noted that all the subjects in Wilson and Niaura's experiment had already participated in a previous experiment (Wilson et al, 1985), which also involved a BPD. Arguably, these subjects might not have entirely believed the placebo manipulations the second time. The present experiment will check Wilson and Niaura's conclusion that belief of having drunk did not affect the voluntary suppression of penile response.

Genital response, perception of it, and subjective sexual arousal are processes that interact in a complex way (Bancroft, 1989). To study this interaction and how it is affected by alcohol, two different measures of subjective response were taken in addition to the actual measurement of penile response. Subjects were asked to report 'how sexually aroused they felt' and 'what degree of erection they reached'.

3.2. Method

3.2.1. Design and subjects

A balanced placebo design (BPD) was used to examine the effects of alcohol and the belief of having drunk alcohol on sexual response under two conditions: (1) while watching an erotic video under instructions to inhibit sexual arousal and (2) during self-induced arousal through imagery. As already explained BPD is a 2 x 2 factorial design where actual content of the drink (alcohol vs. placebo) and information given to the subject (alcoholic vs. soft drink) are manipulated independently yielding four experimental groups:

G-T : given alcohol, subject told the drink contains alcohol.

G-nT : given alcohol, told there is no alcohol in the drink.

nG-T : no alcohol is given, told the drink contains alcohol.

nG-nT : no alcohol is given, told there is no alcohol in the drink.

Fourteen subjects formed the final sample whose responses were analyzed. Seven received an alcoholic beverage, the other seven a soft drink. Four subjects in the former group and three in the latter group were told that their drinks contained alcohol. The rest of the subjects were told that they had been allocated to the 'control group' and that there was no alcohol in their drinks. Subjects were undergraduate and postgraduate students. They were approached personally and no pressure to persuade them to participate was applied with those who did not show immediate willingness. Only those volunteers who were social drinkers (i.e. not teetotallers) were accepted. Additional exclusion criteria were: a history of psychiatric illness, drinking problems, sexual dysfunction, and suffering from any physical condition incompatible with alcohol intake.

Four additional subjects were recruited, but their data were not complete, and therefore were not analyzed, for several reasons. Two of them had been assigned to the group receiving alcohol and had been told that no alcohol was contained in their drinks. Although the manipulation was successful at first, when alcohol started reaching the blood stream both subjects clearly noticed the effects and became fully aware of the deception. Both of them were discharged. In one case, the subject, who was a research psychologist and was aware of this

kind of experimental design, suspected the manipulation and looked for any sign of alcohol intoxication. In the other case, although the subject was not a teetotaller (he did not consider himself to be one and he drank alcohol practically every week), in closer inquiry after his discharge, he reported that he thought of himself as hypersensitive to alcohol. It is not surprising that such a person, who had very rarely had more than a glass of wine, noticed the effects of the equivalent of two pints of beer drunk in fifteen minutes on an empty stomach. Technical problems which made it impossible to measure the erectile response were the causes of the remaining two unsuccessful experimental sessions.

The fourteen final valid subjects had an average age of 26.0 years (ranging from 19 to 40, SD=5.63) and drank a mean of 16.77 units of alcohol per week (SD=11.88). There were two married, three cohabiting, and 11 single subjects.

Subjects were asked to fast for four hours, and not to take any alcohol or any drug or medicine for twelve hours, prior to the experimental session.

3.2.2. Alcohol dose and placebo manipulations

Subjects in the alcohol conditions, irrespective of the information they received about the content of their drinks, were administered a dose of alcohol which was intended to produce a maximum blood alcohol concentration (BAC) of 70%. The dose given to each particular subject was calculated on the basis of the total body water and blood water using the equation given by Watson et al. (1981). This is an updated

version of the classic Widmark equation. Total body water was calculated from anthropometric data through nomograms based on regression equations derived from empirical data (Watson et al., 1980). The average dose subjects received was 0.545 g of ethanol/kg of body weight (SD= 0.019). Ninety per cent pure ethanol was diluted in "Safeway" lemon and lime drink and lime juice at the rate of 1:9:1 (ethanol:lemon and lime drink:lime juice). In the non-alcohol condition subjects drank an amount of plain lemon and lime drink similar to that which would have been necessary to dilute the alcohol dose corresponding to their body weight. For the group who did not receive alcohol and was told the truth about the beverage content, the drink was served directly from a can. For those who drank alcohol and were accurately informed of this, soft drink and alcohol were measured and mixed up in front of the subject. For the group who actually received a non-alcoholic drink but was told that, because they had been assigned to the "experimental group", they were given alcohol, the soft drink was mixed with the content of an "alcohol bottle", which actually contained plain water. The experimenter explained to the subject that the bottle contained 90% pure ethanol and proceeded to mix a precisely measured amount of the content of the bottle with the soft drink. Those who actually drank alcohol, and had been told that the content of their drinks was entirely non-alcoholic, were served their drinks directly from the can of soft drink which was opened in front of the subject. Previously, a tiny hole had been made in the can through which the necessary amount of liquid was extracted by means of a syringe and was replaced by a proportional amount of alcohol and lime juice. In every case, the total consumption time was fifteen minutes. The total dose was divided into five equal parts and the subject was instructed to consume each sub-dose within three minutes,

in order to keep the rate of intake constant for all subject.

3.2.3. Measurements and apparatus

Penile diameter was recorded on a 'Grass' model 7D polygraph by means of a mercury-in-rubber strain gauge (Bancroft, 1974) connected to a Grass 7PIE D.C. preamplifier via a Wheastone Bridge. The penile device was placed on the proximal half by the subject, out of the sight of the experimenter, on the penile shaft. The strain gauge was calibrated before each session using two perspex discs of 25 and 26 mm in diameter to give a ratio of 3 mm of pen deflection per 1 mm of diameter change.

Both after the erotic video and the self-arousing task subjects were asked to report how they felt on a number of Visual Analogue Scales (VAS). They were required to indicate 'how sexually aroused they felt' and 'what degree of erection they experienced' (see Appendix). Affective state was also measured through VASs. Subjects had to mark according to how they felt on nineteen 100 mm long horizontal lines anchored by the words "Not at all" and "Extremely", headed by adjectives referring to different affective states. Eleven of the affective adjectives were similar to the ones used by Heiman (1980). Two VASs headed by the words "Intoxicated" and "Drunk" were intercalated between the other adjectives. (See Appendix)

3.2.4. Procedure

All the experimental sessions took place between 5 pm and 7 pm in

order to control for the possible influence of circadian rhythm on the effect of alcohol on the organism.

On arrival subjects were shown the experimental chamber and the penile transducer. The measuring procedure and what they were supposed to do during the experiment were fully explained to the subjects. Afterwards, subjects were given the opportunity to reconsider their participation. If they definitely agreed to carry on with the experiment they were taken to a nearby room where, after checking the fulfilment of the pre-experimental requirements and that they did not suffer from any medical problem incompatible with taking alcohol, subjects were asked to sign a consent form (see Appendix).

The experimenter brought the drink and proceeded with the appropriate placebo manipulation depending on the group to which the subject had been randomly assigned. The total amount of drink was poured in five glasses and the subjects instructed to consume each one within three minutes.

Twenty minutes after finishing his drink the subject was escorted to the experimental room, where he received precise instructions about what he had to do during the experiment and was then left alone. A checklist to remind him of the successive steps of the experimental procedure was visible. When he had put on the plethysmograph and was sitting in the easy chair, the subject completed the first set of VASs and then pressed a button to indicate to the experimenter that he was ready. Twenty five minutes after the consumption of the drink, a ten-minute neutral video -a documentary - started, which the subjects watched under instructions to look at it and relax. The erotic video

followed immediately after the documentary. It lasted for five minutes and depicted explicit heterosexual interactions including oral sex and intercourse. Subjects had been instructed to 'look at the film and try not to become sexually aroused'. Once the video had finished, subjects answered the VASs and pressed the button to indicate they were finished. Then a three minutes section of the same documentary was shown. The subject was instructed to pay close attention to it and try to memorize everything they heard and saw, because, they were told, recall was going to be tested later. It was thought that requiring them to concentrate in this way would facilitate the return to baseline erectile levels. Following the end of this three minutes neutral video, subjects had to count up to 50 and press the button to indicate that they were starting to try to become aroused. The instructions they had received were to 'try hard to get sexually aroused through imagery'. They were told that they could use any kind of fantasy, including the erotic video previously seen, and that they were not going to be asked about the content of their fantasies. After three minutes they were asked to stop and to go on to complete the scales and subsequently to take the penile transducer off and leave the experimental chamber. Finally, the subject was fully debriefed about the content of the drink he had ingested.

3.3. Results

Due to the small number of subjects in each cell nonparametric statistical techniques were used for data analysis. Meddis (1984) has recently developed an ANOVA by ranks procedure capable of dealing with a variety of designs including factorial designs. The mathematical principles underlying Meddis's approach are similar to those behind most popular nonparametric tests. Roughly speaking, the

Table 3.1. 2 x 2 ANOVAs by ranks on self-reports of intoxication and drunkness.

Phase	Dep. Var.	Source	specific test L	Z	p
(1)	Intoxicated	Alcohol	16	1.02	n.s.
		Belief	49	3.14	0.001
		A x B	-10	0.32	n.s.
	Drunk	Alcohol	22	1.4	n.s.
		Belief	45	2.88	0.002
		A x B	-10	0.32	n.s.
(2)	Intoxicated	Alcohol	14	0.9	n.s.
		Belief	45	2.89	0.002
		A x B	-4	0.7	n.s.
	Drunk	Alcohol	19	1.21	n.s.
		Belief	40	2.56	0.002
		A x B	-14	0.06	n.s.
(3)	Intoxicated	Alcohol	16	0.8	n.s.
		Belief	42	2.71	0.003
		A x B	2	1.1	n.s.
	Drunk	Alcohol	17	1.09	n.s.
		Belief	41	2.63	0.004
		A x B	-9	0.38	n.s.

Coefficients for contrasts, corresponding to the groups G-T, G-nT, nG-T and nG-nT, were: 1,1,-1,-1 for the alcohol factor; 1,-1,1,-1 for the belief factor; and -1,1,1-1 for the interaction. The alternative hypotheses for the specific tests that the coefficientes reflect were that alcohol actually drunk and the belief of having been given an alcoholic drink would increase self-reported feelings of intoxication and drunkness.

Phase 1: 20 minutes after finishing the drink
Phase 2: After watching the erotic video (35-40 minutes after consumption)
Phase 3: After fantasy (50-55 minutes after consumption)

statistics to be used in the analysis of the 2x2 factorial design can be considered as an extension of the well-known Mann-Whitney test for two independent samples. Meddis (1984) provides two solutions for nonspecific (unplanned) comparisons. As there are unequal cell sizes in the present design, the one capable of dealing with the interaction effect in this situation has been chosen. Thus, the statistic 'H' for nonspecific or unplanned tests has been calculated as H=Z x Z, Z being the result of the corresponding specific test. H is distributed as chi-square with (k-1) degrees of freedom. H calculated in this way represents a conservative post hoc test (see Meddis, 1984, p. 291).

3.3.1. Checks of placebo manipulations

Self-reports of perceived intoxication and drunkenness were found to be significantly influenced by the information provided about the content of drinks regardless of the actual composition of these. Results of the 2x2 anova by ranks are shown in table 3.1.. The main effect of the belief of having drunk alcohol was always statistically highly significant. Alcohol actually consumed did not affect subjects' reports of intoxication or drunkenness. However, when, at the end of the experimental session, subjects who had been told that their drinks contained alcohol were asked to report how much they thought they had consumed, those that actually received alcohol said they believed they had drunk the equivalent to 2.25 pints of beer (SD= 0.64), whereas those who received the placebo guessed they had consumed only 1.16 pints (SD=0.28). This difference does not reach statistical significance (Mann-Witney U=0.5) probably due in part to the small sample size.

3.3.2. Psychophysiological and subjective sexual arousal

Baseline measures of penile tumescence were calculated as the average penile diameter over 30 seconds before the start of the erotic stimulation. Measures were taken from the polygraphic trace records (3 mm pen deflection = 1 mm penile diameter) every seven seconds and the five values obtained averaged. A subject in the group that received alcohol and were told so failed to return to baseline levels after the erotic video. He was removed from the sample for the analysis of the self-arousing task data. For the rest of the subjects the differences between penile diameter baselines prior to erotic video and before the imagery task were marginal (Mean=0.5 mm, ranging from -0.7 to 1.5).

The following measures of penile response were taken from the polygraphic records: (1) maximum increase of penile diameter, (2) latency to reach an increase of 3 mm, and (3) latency to reach an increase of 10 mm in penile diameter. When these levels of erection were not reached throughout maximum latency values were recorded as scores (300 or 180 seconds). Only two subjects failed to reach the increase of 10 mm during the voluntary inhibition procedure. In the self-arousing phase 3 subjects had not reached this level of erection after three minutes; two of them (one in the G-nT group and the other in the nG-T group) did not even attain the 3 mm level. It is important to note that, because rank statistics were used, the presence of these cases does not affect the results as much as it would have been the case if parametric statistics had been used.

Voluntary inhibition of sexual arousal

Data of physiological and subjective responses during the voluntary inhibition task are given in table 3.2. 2x2 analyses of variance by ranks on the different measures of sexual responding (Table 3.3.) reveal a statistically significant main effect of alcohol on latency to 10 mm (H=5.29, df=1,p=0.02) and on perceived sexual arousal (H=4.97,df=1,p=0.025), i.e. people who actually drank irrespective of what they were told took less time to reach 10 mm of penile diameter and reported higher levels of sexual arousal. Subjects told that they were having alcoholic drinks regardless of the actual content of these tended to report more sexual arousal, although this effect did not reach statistical significance on the nonspecific -bidirectional- test (H=2.95,df=1,p=0.088). No main or interaction effects were found on max increase of penile tumescence, latency to 3 mm or perceived erection.

Self-arousing through imagery

Results of the 2x2 anovas by ranks for the self-arousal task are shown in table 3.5. (descriptive statistics are given in table 3.4.). Alcohol significantly affected latency to 10 mm (H=4.04,df=1,p=0.045) . Interestingly, this effect was in the opposite direction to the one found during the voluntary inhibition of arousal. While trying to get sexually aroused through imagery subjects who had actually consumed alcohol irrespective of their beliefs about the content of their drinks took significantly longer to reach 10 mm increase in penile diameter. Subjects who received alcohol also

Table 3.2. Voluntary inhibition task. Means, medians and standard deviations.

Dep Var	group	Mean	Median	St Dev	n
MAX	G	14.80	14.00	3.07	7
INCREASE	nG	14.42	13.50	5.27	7
(mm)	T	14.30	14.00	4.22	7
	nT	14.92	13.50	4.38	7
LATENCY	G	7.75	7.30	6.22	7
to 3 mm	nG	11.42	13.90	5.50	7
(sec)	T	8.55	7.30	5.58	7
	nT	10.62	9.40	6.58	7
LATENCY	G	11.74	10.80	7.05	7
to 10 mm	nG	21.51	20.10	7.83	7
(sec)	T	12.97	10.90	8.45	7
	nT	20.28	20.10	8.01	7
PERCEIVED	G	76.42	77.00	3.04	7
SEXUAL	nG	55.28	52.00	20.19	7
AROUSAL	T	74.00	75.00	12.32	7
(100-mm VAS)	nT	57.71	61.00	19.26	7
PERCEIVED	G	58.00	57.00	28.24	7
ERECTION	nG	41.00	36.00	19.18	7
(100-mm VAS)	T	57.42	57.00	28.94	7
	nT	41.57	36.00	18.71	7

G: given alcohol
nG: no alcohol given
T: told drink contains alcohol
nT: told drink contains no alcohol

Table 3.3. Voluntary inhibition task. 2x2 ANOVAs by ranks.

		specific test			non spec. test		
Dep Var	**Source**	**L**	**Z**	**p**	**df**	**H**	**p**
MAX	**Alcohol**	4	<1	n.s.			
INCREASE	**Belief**	-4	<1	n.s.			
ERECTION	**A x B**	-15	<1	n.s.			
LATENCY	**Alcohol**	17	1.08	n.s.			
to 3 mm	**Belief**	9	<1	n.s.			
	A x B	3	1.16	n.s.			
LATENCY	**Alcohol**	36	2.3	.01	1	5.29	.02
to 10 mm	**Belief**	24	1.53	.06			
	A x B	-5	<1				
PERCEIVED	**Alcohol**	35	2.23	.012	1	4.97	.025
SEXUAL	**Belief**	27	1.72	.04	1	2.95	.088
AROUSAL	**A x B**	-1	<1				
PERCEIVED	**Alcohol**	23	1.47	.07			
ERECTION	**Belief**	21	<1	n.s.			
	A x B	-23	<1	n.s.			

Coefficients for contrasts, corresponding to the groups G-T, G-nT, nG-T and nG-nT, for the variables 'max increase erection', 'perceived sexual arousal' and 'perceived erection', were: 1,1,-1,-1 for the alcohol factor; 1,-1,1,-1 for the belief factor; and -1,1,1-1 for the interaction. For the latency variables the coefficients were: -1,-1,1,1 for the alcohol factor; -1,1-1,1 for the belief factor; and -1,1,1,-1 for the interaction.

Aternative hypotheses for specific tests reflected on the coefficients for contrasts were that alcohol actually drunk and the belief having been given alcohol would increase 'max increase erection' and both subjective measures, while diminishing latency measures.

Table 3.4. Self-arousing task. Means, medians and standard deviations.

Dep Var	group	Mean	Median	St Dev	n
MAX	G	8.70	10.30	4.03	6
INCREASE	nG	13.52	14.50	6.74	7
(mm)	T	11.50	10.50	6.92	6
	nT	11.12	11.00	5.63	7
LATENCY	G	8.88	9.30	6.35	6
to 3 mm	nG	6.51	4.60	5.30	7
(sec)	T	8.21	6.85	6.37	6
	nT	7.08	4.60	5.49	7
LATENCY	G	14.98	14.95	2.88	6
to 10 mm	nG	9.45	7.80	5.08	7
(sec)	T	12.68	14.55	5.89	6
	nT	11.42	10.40	4.41	7
PERCEIVED	G	61.00	56.00	19.30	6
SEXUAL	nG	62.57	69.00	23.21	7
AROUSAL	T	60.00	62.00	17.73	6
(100-mm VAS)	nT	63.57	62.00	24.31	7
PERCEIVED	G	31.16	27.50	21.09	6
ERECTION	nG	47.50	64.50	29.00	7
(100-mm VAS)	T	43.33	44.50	28.20	6
	nT	38.33	39.00	26.03	7

G: given alcohol
nG: no alcohol given
T: told drink contains alcohol
nT: told drink contains no alcohol

Table 3.5. Self-arousing task. 2 x 2 ANOVAs by ranks.

		specific test			non spec. test		
Dep Var	**Source**	**L**	**Z**	**p**	**df**	**H**	**p**
MAX	**Alcohol**	34	1.93	.026	1	3.72	0.054
INCREASE	**Belief**	4	<1	n.s.			
ERECTION	**A x B**	-15	<1	n.s.			
LATENCY	**Alcohol**	0	<1	n.s.			
to 3 mm	**Belief**	-4	<1	n.s.			
	A x B	3	<1	n.s.			
LATENCY	**Alcohol**	21	2.01	.021	1	4.04	.045
to 10 mm	**Belief**	-1	<1	n.s.			
	A x B	-5	<1				
PERCEIVED	**Alcohol**	8	<1	n.s.			
SEXUAL	**Belief**	10	<1	n.s.			
AROUSAL	**A x B**	13	<1	n.s.			
PERCEIVED	**Alcohol**	15	1.2	.114			
ERECTION	**Belief**	-1	<1	n.s.			
	A x B	4	<1	n.s.			

Coefficients for contrasts, corresponding to the groups G-T, G-nT, nG-T and nG-nT, for the variables 'max increase erection', 'perceived sexual arousal' and 'perceived erection', were: 1,1,-1,-1 for the alcohol factor; 1,-1,1,-1 for the belief factor; and -1,1,1-1 for the interaction. For the latency variables the coefficients were: -1,-1,1,1 for the alcohol factor; -1,1-1,1 for the belief factor; and -1,1,1,-1 for the interaction.

Aternative hypotheses for specific tests reflected on the coefficients for contrasts were that alcohol actually drunk and the belief having been given alcohol would diminish 'max increase erection' and both subjective measures, while increasing latency measures.

showed smaller maximum increases in penile tumescence, although this effect only approached statistical significance (H=3.72, df=1, p=0.054). No other effect was found.

Affective states

2x2 anovas by ranks were performed on every dimension of the three reports of subjective feeling: immediately after settling in the experimental room and before and after the two experimental procedures.

(1) On the first report (approximately 20 minutes after finishing the drinks) those subjects who thought they had drank alcohol reported feeling much more "carefree" (Mean difference= 33.85; H=7.18, df=1, p=0.007). This was the only statistically significant result, although some trends should be noted. Subjects who believed they had consumed an alcoholic drink tended to feel more "excited" (H=2.75, df=1, p=0.10) and more "interested" (H=3.43, df=1,p=0.06). Those who actually drank alcohol tended to feel more "excited" (H=2.75, df=1, p=0.10), less "bored" (H=2.97, df=1, p=0.088) and more "interested" (H=2.75,df=1,p=0.10).

(2) After the inhibition task, subjects who believed they were under the effects of alcohol felt significantly less "disgusted" (H=4.73, df=1, p=0.03), less "embarrassed" (H=3.96, df=1, p=0.048) and less "nervous" (H=3.92,df=1,p=0.048). They also reported feeling less "uncomfortable", although this effect did not reach statistical significance (H=3.68,df=1,p=0.057). Actual intoxication made subjects feel less "guilty" (H=4.41,df=1,p=0.035). These same subjects tended to feel less

"uneasy", although this effect only approached statistical significance (H=3.42,df=1,p=0.065). There was an interaction effect on reports of "excitation" (H=4.28, df=1, p=0.04). Further inspection of the data revealed that the belief of having drunk a soft drink made subjects feel less "excited" only when no alcohol had been consumed. Means for G-T, G-nT, and nG-T groups were virtually identical (respectively, 61.25, 72 and 70), whereas the mean for the nG-nT group mean was notably lower (Mean=23.75).

(3) During the self-arousing phase those who actually drank alcohol reported feeling more "carefree" (H=4.97, df=1, p=0.026). No other significant effect was found.

3.3.3. Intercorrelations among dependent variables

Correlation coefficients (Spearman's rho) between ratings of 'perceived erection' and 'perceived sexual arousal' did not reach statistical significance in either experimental task. During the voluntary inhibition procedure a Spearman's rho of 0.50 (p=0.066) was found, which, it could be argued, reflects a tendency that could have been statistically significant if a larger sample had been used. A negligible rho of 0.27 (n.s.) was found during the fantasy phase.

In order to explore the relationship between physiological indices of sexual responding and subjective estimates and how experimental factors influenced this relationship, nonparametric Spearman's rho correlation coefficients were computed between physiological parameters and subjective ratings within the four different groupings corresponding to the main effects of the two experimental factors (Table 3.6.).

Table 3.6. Spearman's Rho correlation coefficients between indices of physiological and subjective sexual response.

	Voluntary inhibition		Self-arousing task	
	perceived sexual arousal	perceived erection	perceived sexual arousal	perceived erection
MAX INCREASE ERECTION				
G	.09	.35	.69 (.12)	-.13
nG	-.16	.92 (.002)	-.03	.60
T	.32	.50	.82 (.04)	.69
nT	.14	.79 (.032)	-.25	.25
LAT TO 3 MM				
G	-.09	.32	.20	-.23
nG	-.10	-.34	-.35	-.37
T	.14	-.14	-.08	-.57
nT	-.25	.45	-.67 (.09)	.20
LAT TO 10 MM				
G	-.14	.14	-.63	-.08
nG	.12	-.83 (.02)	.03	-.60
T	-.09	-.30	-.63	-.80 (.052)
nT	-.36	-.14	.00	-.14

p values (in brackets) are reported only if less than 0.1 or approaching this level

From the inspection of Table 3.6., it seems that 'lat to 3 mm' did not influence subjective estimates at all. Ratings of 'perceived sexual arousal' do not appear to depend upon actual erectile response to any appreciable extent. On the other hand, the relationship between 'max increase erection' and 'lat to 10 mm' with 'perceived erection' seems to be influenced by the experimental variables (alcohol and belief). When subjects did not drink alcohol the correlation between these two physiological parameters and estimates of the degree of erection attained were high and statistically significant (rho=0.92, p=0.002, for 'max increase erection' and rho=-0.83, p=0.02, for 'lat to 10 mm'). A high correlation between 'max increase erection' and 'perceived erection' was also found when subjects were told that their drinks did not contain any alcohol(rho=0.79, p=0.032).

The pattern, if any, is quite different for the results of the self-arousing task (see table 3.6.). The number of correlations computed and the modest levels of statistical significance obtained lead one to consider that it would be foolhardy to force any post hoc speculative explanation into these results.

3.4. Discussion and conclusions

In the present study, as in Wilson and Niaura (1984), alcohol was found to exert an arousing effect when subjects were asked to inhibit their sexual response to erotic stimuli. Since this positive effect has not been found in other studies where this instructional set was not used (on the contrary, a suppressing effect has been normally found at the level of dosage used here), one can arguably conclude that the action of alcohol in this and Wilson and Niaura's studies was that of impairing

voluntary inhibition of ongoing sexual response. At variance with Wilson and Niaura (1984), however, we did not find a statistically significant effect on latency to 3 mm but on latency to 10 mm of erection increase. Differences in the stimulus material may account for this discrepancy. One could argue that, as stimulation in the present study was more powerful (e.g. explicit erotic film vs. narratives), a ceiling effect occurred: since nonintoxicated subjects were not very successful at inhibiting their responses below 3 mm. On measures of latency to 10 mm of penile diameter change, once the ceiling effect was overcome, the disinhibitory effect of alcohol became apparent. In any case, on examining Table 3.2. a trend for subjects who received alcohol to show shorter latencies to 3 mm of erection increase can be observed, although this difference was clearly nonsignificant statistically.

As expected, when asked to become sexually aroused using fantasy, those who actually drank alcohol showed lower genital sexual response as reflected in an increased latency to reach 10 mm of erection change and a lower 'max increase erection', although the latter effect was statistically only marginally significant. Thus, the arousing effect of alcohol was specific to the situation of voluntary inhibition of sexual response.

The mechanisms through which alcohol acted are not clear. It might be argued that the influence of alcohol on both experimental tasks was via an impairment of the cognitive processes necessary to control (i.e. to supress or to generate) the physiological sexual reaction. One may alternatively argue that the inhibitory influence of alcohol on self-induced sexual arousal is nothing but the result of the well-known physiologically-mediated reducing effect of ethanol on genital sexual

responding, and that the effect of alcohol intoxication on the voluntary inhibition task was of cognitive nature. It would have required a third experimental task, namely, the uninfluenced exposure to erotic stimuli, to help differentiate between these two alternative hypotheses.

Even though the idea of alcohol impairing the cognitive functioning necessary to effectively control (i.e. inhibit) the sexual response has considerable appeal, the correlation data in this study suggest that other explanatory mechanisms should be considered. One might tentatively put forward the hypothesis that alcohol impairs or distorts the perception of the erectile response. Visceral feedback plays an important role in emotion. Additionally, it has been suggested that individual differences in awareness of visceral activity may determine the attainment of voluntary visceral control via biofeedback training (Brener, 1977). We know that in normal circumstances males are highly aware of their penile response: high correlations between physiological response and subjective estimates of sexual arousal are usually obtained (Bancroft, 1974, Geer, 1980). Perception of genital responses has been posited as an important factor in determining subsequent sexual feelings and behaviour (Wallen, 1980). One might suppose that a lack of adequate feedback would impair the control over evoked sexual arousal. In Table 3.6., it can be seen that during the inhibition task the correlation between the physiological parameters and perceived erection was high and statistically significant for those subjects who did not drink alcohol irrespective of the information they received. With the only exception of the correlation between max increase erection and perceived erection when subjects were told that their drinks did not contain alcohol, the rest of correlations were statistically nonsignificant. This supports the hypothesis that alcohol impaired feedback of erectile

response. A moderate correlation of 0.5 between 'perceived erection' and 'perceived sexual arousal' supports the idea of subjective sexual arousal as a complex state based on a number of physiological responses amongst which penile erection is the most specific but not the only one (Bancroft, 1974). 'Perceived sexual arousal' does not appear to be dependent on any index of genital performance at all, whereas 'perceived erection' seems to be strongly determined by 'max increase erection' and 'lat to 10 mm' as long as alcohol has not been ingested. The analysis of the relationships between physiological parameters and subjective estimates and the influence of the experimental variables (alcohol and belief of having drunk) on them has been carried out here as an example of the sort of analysis that may prove to be revealing if performed on larger numbers of subjects. Given the sample size in this experiment the likelihood of Type II error is large and it is impossible to analyze the effects of all the experimental variables and their interaction on the correlations between the different dependent variables properly.

Both alcohol and the belief of having drunk alcohol had a positive effect on general mood, before and after the erotic stimulation. Although not much is known about the effects of mood on sexual response, it is likely that positive mood would facilitate sexual arousal (Bancroft, 1989).

At variance with Wilson and Niaura (1984) the belief of having drunk alcohol increased sexual response during the voluntary inhibition task. One may argue that it would not be very easy to implement the BPD with subjects that have already taken part in a study involving the same design. It has been shown that subjects tend to hide their

suspicion of the deception (Knight et al, 1986). It is likely that the expectancy manipulation in the Wilson and Niaura (1984) study was not totally successful.

Placebo manipulations were successful with the fourteen subjects whose data entered the final analyses in that reports of intoxication and drunkenness were significantly determined by the information given by the experimenter. However, the two groups that were told that they were drinking alcohol differed in the amount of alcohol they estimated they had consumed, with those who actually received ethanol estimating larger doses. This suggests that the subjective state of drunkenness is not an all-or-none phenomenon but one that can vary in intensity and it is a function of diverse sources of information (bodily feedback, expectancies, etc.).

In summary, the results of this experiment confirm that alcohol impairs the voluntary control of ongoing sexual response. An impairment of a cognitive nature is a possible cause of this effect. Data from the experiment also suggest that a distortion of the feedback from the penile response may also contribute to the observed effect of alcohol.

Chapter 4.
ALCOHOL AND STRESS

4.1. Introduction

There exists the widespread popular belief that ethanol has antianxiety properties. This view is also shared by scientists and health professionals. In a report on alcohol and alcoholism by the Royal College of Psychiatrists it can be read: "Even the most casual drinker is familiar with the feelings of warmth, the heightened perception, and the relief from anxiety and stress that alcohol, in reasonable amounts, can endow. Its tranquillizing effect is well-known among the elderly, the lonely, the sad and depressed, and sufferers from chronic pain." (Royal College of Psychiatrists, 1986, p. 46). However, several decades of scientific research on the effects of alcohol on anxiety (or stress) have not produced clear-cut results. On trying to summarize a session of presentations during a recent conference entirely devoted to the topic of 'Stress and Alcohol Use' Nathan (1983) concluded: "it is clear that alcohol increases stress, that it decreases stress, that it does both, and that it does neither". In this chapter I will attempt to give an account of the research into the effects of alcohol on stress over the last few decades. The focus will be on human research, and, in particular, on those studies which have examined the effects of alcohol on the response to experimentally-induced stress after drinking.

The investigation of the effects of alcohol on stress or anxiety has been dominated by the 'tension reducing theory' of alcohol use and abuse - known as Tension Reduction Theory (TRT) or Tension Reduction Hypothesis (TRH). Although numerous precedents can be found, the

first formal enunciation of the TRT is attributed to Conger (1956). Conger was very much influenced by Masserman's animal studies (Masserman and Yum, 1945), in which it was found that cats subjected to an approach-avoidance conflict situation ('experimental neurosis') were more motivated to drink. Based also on his own work with rats and, on the theoretical side, on the neobehaviouristic learning theory of the 40s (e.g. Hull, 1943), Conger (1956) formulated his "drive-reduction hypothesis" of drinking motivation, according to which "the drinking response is learned because it leads to a reduction in drive" (p. 296). As Sher (1987) has rightly pointed out Conger "did not view alcohol as a simple tension-reductor robust across individuals and situations" (p. 228). Conger's explanation of the effects of alcohol was confined to those cases in which a 'drive' is generated by an approach-avoidance situation. Sher (1987) observes that Conger (1956) "explicitly stated that alcohol can produce conflict and increase anxiety" (p. 228) in certain situations in which alcohol by reducing restraining tendencies is the cause of a conflict. However, it has not been the original Conger's formulation the one that has inspired the myriads of experiments testing the tranquillizing effects of alcohol carried out over the last three decades, but the simpler view of alcohol as a general tension reducer, which has often been wrongly attributed to Conger. What most studies have tried to test is the popular and pervasive belief that alcohol has tranquillising effects, not the more specific Conger's formulation of the TRH.

Recently, Sher (1987) has produced a version (a "modest, pared-down version", Sher (1987) has explained) of the TRH: the Stress Response Dampening (SRD) model. In this model, "alcohol is posited to dampen stress response and thus is seen as being particularly reinforcing when

it is consumed in a stressful context" (p. 234). Like the TRH, the SRD model has a corollary: "Because of this, individuals who experience SRD effects are likely to drink with increased frequency and possibly in greater quantity when stressed" (Sher, 1987, p. 234). Sher's formulation expresses the implicit hypotheses of most studies in this field better than the original Conger's TRH. The present review will be mainly concerned with those studies examining the stress-reducing effects of drinking, that is, the first part of the SRD hypothesis. In this field, the terms 'anxiety', 'tension' and 'stress' have been used "roughly synonymously" (Wilson, 1988). Attempts to define these concepts have been scarce.

Spielberger's definition of anxiety describes well the state that most studies in this field have tried to measure. Spielberger (1972) defines anxiety as "unpleasant, consciously-perceived feelings of tension and apprehension, with associated activation or arousal of the autonomic nervous system" (p. 29). Thus, all studies in this field have recorded self-reported stress (or tension or anxiety), and many have included measures of autonomic activity (usually heart rate and skin conductance).

4.2. Survey studies

Besides the experiments investigating the relationship between drinking and stress, there are a number of studies which have been carried out with the purpose of examining people's beliefs concerning the tension-reducing effects of alcohol. Studies in which expectancies about the consequences of drinking have been measured have found that one of the main expected effects of alcohol is tension reduction (Brown et al,

1980). It has also been found that inexperienced adolescents expect alcohol to 'promote relaxation or tension reduction' (Christiensen et al, 1983), which seems to indicate that these expectancies are acquired prior to actual experience with alcohol.

Farber et al (1980) studied the self-reported reasons for drinking of a group of drinkers of varied level of consumption, using factor analysis. Two main factors were identified: 'social drinking' seeking positive reinforcement, and 'escape drinking' to achieve negative reinforcement through the relief of an unpleasant affective state. High levels of alcohol intake were associated with 'escape drinking'. Problem drinkers at large (93%) reported drinking to avert negative emotional states and to relax when distressed. A large number of drinkers (particularly moderate drinkers) seem to drink in order to achieve positive aims. This has been confirmed in studies conducted with college students, in which it has been found that the enhancing of positive experiences is as important a reason to drink as the relief of distress (e.g. Russell and Bond, 1979).

4.3. Stress as the cause of drinking

Anecdotal and retrospective reports suggest that relapse in treated problem drinkers tends to happen when the patients have to face stressful life situations (Marlatt, 1983). Marlatt undertook the experimental investigation of the hypothesis that stress can cause drinking. In the first experiment (Higgins and Marlatt, 1973), stress was provoked in two groups: social and problem drinkers. All subjects participated in a taste rating task and were allowed to drink as much as they wanted. Half of them had been told that after the rating task

they were going to receive painful electric shocks. The shocks were never delivered. Problem drinkers drank more than social drinkers, but the threat of the shock did not generate any difference in consumption in either group. In a second experiment (Higgins and Marlatt, 1975) a social stressor was used with a group of social drinkers. As in the first study, all subjects participated in a task in which they were asked to compare and to rate the taste of various alcoholic drinks, and in which they could drink without restrictions. Half of the participants had been told that after the tasting session they would be in a situation in which a group of attractive females would rate them on several dimensions including attractiveness. The group expecting to be assessed drank significantly more. Marlatt (1983) has argued that the "explanation for the discrepant findings in the two studies is that fear of physical pain is not a meaningful source of tension as it relates to the consumption of alcohol", because "people do not expect that alcohol will have any tension-reducing effects in this specific situation" (p. 281).

Other experiments investigating stress-induced drinking have found mixed results. Tucker et al (1980) used a difficult intellectual task, presented as an intelligence test, to cause stress. The subjects who performed in this task drank more than those who were given an easier test. However, Phil and Yankofsky (1979) found results in the opposite direction in an experiment in which the subjects were administered a bogus intelligence test, and in which half of them received failure feedback while the other half were given success feedback. Against what was expected, the first group drank less, although they reported feeling more depressed and anxious.

It has been found that stress provokes less drinking if some sort of

coping opportunity is provided. Marlatt et al (1975) provoked stress by having the subjects evaluated negatively. Those who were given the opportunity to retaliate against the person who had produced the negative evaluation drank less than those who were not allowed revenge. Stricker et al (1975) found that the threat of an evaluative public speaking task made people drink more, but that giving relaxation instructions reduced this effect.

In rats, different stressors, physical (e.g. footshock, immobilization) and psychological (e.g. submissiveness) have been shown to increase the intake of ethanol (Pohorecky, 1990).

4.4. Effects of alcohol on mood

A large number of studies have looked at the affective effects of drinking by comparing mood before and after drinking either a placebo or an alcoholic beverage. Lang (1983) has summarized this literature in this way: "Overall the consensus is that drinking does something to mood, but just what that something is may vary with subject gender, alcohol dose, drinking experience, rate of intoxication, etc. This ambiguity appears to underscore the role of psychosocial variables in alcohol's effects, while fortifying the apparent legitimacy of attributing almost any performance outcome, at least indirectly, to the influence of drinking on affect. " (p. 236). Sher (1987) fully agrees: "The affective consequences of alcohol consumption appear inconsistent, with some studies finding increased positive affect, other studies negative affective consequences, and still other studies concomitant negative and positive mood changes." (p. 229). Sher (1987) thinks that the reason for these contradictory results is that the effects of alcohol on mood are "closely

dependent upon the context in which drinking occurs" (p. 230).

Cappell and Greeley (1987), however, totally disagree with Lang's and Sher's view, and affirm that "there appears to be excellent consistency in this literature" (p. 39). This disagreement illustrates a feature of the research on the TRH. It is not difficult to find a substantial number of studies in support of the hypothesis, although negative results are never scarce. Thus, a review of the literature focusing on the positive results, a practice to a certain extent legitimate in science (Sternberg, 1988), will lead to an optimistic conclusion. Tucker et al (1982) have succeeded in expressing the implications of the confusing results of the long list of studies that they reviewed looking at mood after drinking by concluding that "the possibility of tension-reducing effect of alcohol is not eliminated by current evidence" (p. 173). Tucker et al (1982) also point out a methodological flaw in these studies, i.e. the almost exclusive use of measures of negative affect, and emphasize the necessity of using dependent measures of both positive and negative mood. This bias towards negative affect has appeared also in the interpretation of the physiological effects of alcohol (see below).

4.5. Effects of alcohol on the ANS

The effects of alcohol on the Autonomic Nervous System (ANS) are of special interest for various reasons. Firstly, ANS plays a crucial role in the emotional processes (Sartory and Lader, 1981). On the other hand, ANS-dependent indices (mainly Heart Rate and Electrodermal Activity) have been used as dependent variables in many studies looking at the effects of alcohol on stress.

The increase of HR following the consumption of ethanol has been a very consistent finding. Naitoh (1972), in his review of the psychophysiological effects of alcohol, concluded that "the heart rate increase due to alcohol has been found to be very reliable and replicable from one study to another." (p. 414). Alcohol has also been found to increase skin blood flow (Gillespie, 1967, cit. in Naitoh, 1972). Both 'in vitro' studies and research on intact animals have shown a depressive effect of alcohol on cardiac contractility (Knott and Beard, 1972).

(Electrodermal Activity)

The effects of alcohol on EDA have not been found to be consistently in one direction or another. Alcohol has been found to reduce skin conductance responses (Lienert and Traxel, 1959, Carpenter, 1957). Carpenter and Greenberg (1957) found that alcohol reduced skin conductance level (SCL), but an increase in SCL after alcohol consumption has also been reported (Jones et al, 1976). The results of a study by McDonell and Carpenter (1959) illustrate well the importance of context. They found that drinking in a group increased SCL but drinking alone did not.

Alcohol has also been found to reduce muscle tension (Schuckit et al, 1981, Steffen et al, 1974).

Alcohol seems to elicit a stress-like reaction with release of adrenocorticotropic hormone (Van Thiel, 1983). Eisenhofer et al (1986) found increased baseline levels of plasma adrenaline after the consumption of a dose of alcohol of 0.8 g/kg.

On the basis of the effects of alcohol on the ANS it is impossible to

conclude whether ethanol is relaxing or arousing (Sher, 1987). On the other hand, it is necessary to distinguish between the physiological effects of alcohol on the ANS and the psychological significance of these changes. Autonomic changes may be caused directly by alcohol, and in that case they would not have any significance as to the psychological state of the organism, the same as an increase in HR caused by a change in posture from lying to standing does not have any psychological meaning. Studies in which physiological parameters have been measured after drinking, without any attempt to manipulate or control the psychological context, cannot shed light upon the psychological significance of the physiological changes observed. There has been a tendency in these studies to interpret any increase in arousal as an indication of stress, which is not necessarily true.

4.6. Effects of alcohol on stress-response

The most important group of studies analyzing the relationship between alcohol and stress is that in which the influence of alcohol on the response to a stressing situation is examined. Subjects in these experiments (animals or humans) are given a certain dose of alcohol and then exposed to a stressor while their response is measured, often including indices of ANS activity.

4.6.1. Animal research

In a classic review of the research on the effects of alcohol on stress in animals, Cappell (Cappell and Herman, 1972) concluded that the evidence was inconclusive. Hodgson et al (1979) challenged this conclusion and, after reviewing the evidence again, concluded that

alcohol did have an anxiolytic effect if anxiety was operationalized as 'fear' (understood as the expectation of the occurrence of an unpleasant event), or as 'frustration'(as generated by the nonoccurrence of an expected pleasant event). The failures to demonstrate an effect of alcohol happened, Hodgson et al (1979) argued, when the paradigms of escape and active avoidance had been used. These behaviours, once established, are not motivated by fear, and, hence, are not an appropriate test of the tension reduction hypothesis, Hodgson et al (1979) claimed.

Pohorecky (1990) has recently reviewed animal research on the interaction of ethanol and stress with special emphasis on physiological measurement. Alcohol influences numerous physiological consequences of stress. Ethanol reduces the decrease of noradrenaline level and turnover, and the reduction in 5-hydroxytryptamine levels and alpha-1-receptor binding, that stress causes. Also the changes in the brain in beta-endorphins and GABA that stress induces are influenced by ethanol. Alcohol also reduces the stress-induced increases in plasma catecholamines, corticosterone, non-sterified fatty acids and aminoacids, and antagonizes the decline in adrenal catecholamines.

As a whole animal research examining the effects of alcohol on stress response is supportive of the TRH.

4.6.2. Human research

A number of studies have examined the effects of alcohol on the human stress response elicited by noxious stimuli, usually electric shocks.

Lindman (reported in Lindman, 1983) compared the performance of a group of moderate social drinkers when sober and after drinking 0.9 g/kg of ethanol in a task in which the subject had to cross a floor and then reach a small ball floating on the surface of a water bowl, under the risk of receiving a 'marginally unpleasant' shock when the finger touched the surface of the water. There were different probabilities of occurrence of the shock. Time spent in the different parts of the task was taken and the movement of the hand was filmed. The variable threat resulted in significant differences in speed of movements (the more probable the shock was the slower the movements were), but this difference did not exist when alcohol was consumed. When alcohol was drunk the movements were significantly faster and there were fewer hesitation episodes, although skin conductance level was not affected.

Dengerink and Fagan (1978) also used an electric shock as stressor, with a population of male undergraduates. Two doses (0.55 and 0.95 g/kg) were compared against placebo in a between subjects design. Both doses of alcohol increased self-reported anxiety, heart rate and skin conductance (level and response), in response to the stressor. Alcohol did not influence tolerance to the shocks.

Polivy et al (1976) tested female subjects using a reaction time task in which they were told to expect a "painful but not dangerous" electric shock. A dose of 0.48 g/kg was used in the context of a BPD, in which half of the subjects were told that the drink administered was vitamin C. Alcohol reduced self-reported anxiety. However, the induced belief that alcohol was consumed increased anxiety.

Sutker et al (1982) also utilized a BPD (with a dose of 0.63 g/kg) in an experiment in which males and females were exposed to a situation of fear to electric shocks, and in which psychophysiological and self-report measures of tension were taken. The results did not reveal any clear effect of alcohol or instructional set.

In a large study, Levenson et al (1980) tested ninety-six male social drinkers in two different stressing tasks (self-disclosuring speech and threat of electric shock) in a between subjects design in which a BPD was nested. They used a high dose of 1 g/kg. Dependent measures included heart rate (HR), skin conductance level (SCL), pulse transmission time (PTT) and self-reported anxiety. Alcohol per se significantly reduced stress-induced increase of HR, PTT and self-reported anxiety, in a comparable way for both stressors. Induced belief of alcohol consumption did not have any effect.

Some ingenious procedures have been used to provoke anxiety. Lindman (reported in Lindman, 1983) asked a group of female volunteers who were, to some extent, fearful of mice although not phobic, to transfer a laboratory mouse from one jar to another. Half of the subjects drank 0.9 g/kg. Alcohol reduced hesitation behaviour and self-reported anxiety. Rimm et al (1981) used a similar procedure but a bigger animal. Subjects in this experiment were asked to approach a 4-foot boa constrictor. A dose of ethanol of 0.5 g/kg reduced self-reported fear but did not affect approach behaviour. The reason for the discrepancy between these results and Lindman's might lie in the lower dose used by Rimm et al. Although one could argue that it is unlikely that 0.9 g/kg of ethanol, or even a bigger dose, could ever make reluctant volunteers approach such a snake.

Thyer and Curtis (1984) asked 22 severe phobic patients to approach their phobic animal on two occasions: first, sober, and, later, after drinking a dose of 0.8 cc of 100% proof vodka/lb of body weight. Alcohol intoxication did not reduce any of the measures of anxiety (subjective fear, behavioural avoidance, HR, BP and perceived autonomic arousal).

Steele et al (1981) examined, in a series of experiments, the interaction between alcohol and the state of cognitive dissonance generated by asking university student subjects, who were, not surprisingly, opposed to an increase in tuition fees, to write an essay in favour of the fees increase. According to the theory of cognitive dissonance, to reduce the state of dissonance, which is experienced as an unpleasant affective state, people change attitudes becoming more consistent with the imposed behaviour. Thus, in this study subjects would tend to adopt an attitude more favourable to the increase in tuition fees, in order to reduce the unpleasant dissonant state. The change of attitude can be used as a measure of the tension generated by the dissonance. Steele et al (1981) found that the dissonant state did not affect the amount of alcohol consumed in a taste rating task conducted after the dissonance had been induced. When the subjects performed the taste rating task before the dissonance procedure, "whatever drinking occurred was sufficient to eliminate dissonance-reducing attitude change" (p. 831). The amount of drink that subjects consumed in the taste rating task was not measured, but it could never have exceeded the total amount of drink provided, which was 4 oz of vodka. As no placebo control was included it is impossible to ascribe the dissonance-reducing effects of drinking to the pharmacological properties of ethanol.

In Logue et al (1978) the tension-inducing situation was a test of driving skills that male and female subjects performed after drinking one of three different doses (0.5 g/kg, 0.8 g/kg or 1.2 g/kg). Self-reported anxiety increased in a dose-related fashion.

Using a within-subject design, Eisenhofer et al (1986) investigated the effects of a dose of 0.8 g/kg on the psychophysiological response to stress produced by two tasks: a cognitive task which involved the threat of electric shocks, and a competitive electronic game. Alcohol reduced the systolic blood pressure increase in both tasks; diastolic blood pressure was reduced only in the electronic game task. Alcohol reduced cardiac acceleration and the increase in plasma adrenaline during the cognitive task, but not during the electronic game.

Marlatt (1983) suggested a fundamental difference between social and non-social stressors in terms of the implications for the effects of alcohol on the stress response by them elicited. A number of experiments have examined the effects of alcohol on the tension produced by social stressors. The procedure most commonly used to elicit social stress has been one in which subjects are asked to deliver a speech for few minutes, usually to a confederate of the opposite sex, talking about themselves. This procedure of social stress induction, which has usually been called 'self-disclosure' speech, was first used to study the effects of alcohol by Wilson and Abrams (1977), who adapted it from Borkovec et al (1974). It has been claimed that repeated administration of this task does not produce habituation of the stress response (Abrams, 1983).

Wilson and Abrams (1977) employed a BPD to test a group of male undergraduates. All men were asked to interact with a female confederate and try to make a favourable impression. Subjects allocated to one of the alcohol cells of the BPD drank 0.5 g/kg. Those who were induced to believe that they had drunk an alcoholic beverage showed smaller HR increase during the stressful interaction. Self-reported anxiety (State-Trait Anxiety Inventory) was lower in those who believed that they had drunk alcohol, although the difference was only a statistically non-significant trend. The videotapes of the interaction were rated on several dimensions. Those who thought they had consumed alcohol were rated as less inhibited, more relaxed, and more dominant. Alcohol did not have any effect.

Using similar design and set-up, Abrams and Wilson (1979) tested the stress-reducing effects of alcohol and the belief of having consumed alcohol on a group of females who interacted with a male confederate. Those who believed that they had been drinking alcohol had larger increases in HR and their peers rated them as more anxious. Alcohol content of the drinks did not affect any of the dependent variables.

Keaman and Lisman (1980) conducted two related studies to examine the effects of alcohol on 'heterosexual social anxiety' in socially anxious males and in normal male undergraduates. A 2 x 2 design was used in which one factor was drink content (alcohol vs placebo) and the other the instructional set (negative vs positive effects of alcohol). In the first study, a sample of men suffering from social anxiety was studied using a dose of 0.4 g/kg and a stressor similar to that of Wilson and Abrams (1977). The shy men who consumed alcohol performed less skilfully in the social interaction: spent less time speaking and paused more

frequently, tried to pass the responsibility of the conversation to the female confederate, needed a prompt from the confederate to reinitiate the conversation more frequently, and a greater proportion of them forgot the name of the confederate. However, the ratings that the 'alcohol subjects' gave on their own performance were not worse than those of the placebo group. Those subjects who drank alcohol reported a larger number of negative self-statements than subjects in the placebo group. Alcohol did not affect subjective anxiety. Those who received the negative instructional set regarding the effects of alcohol on performance reported increased anxiety before and after the interaction. Drink content did not affect subjective anxiety. Neither experimental variable affected the physiological response (HR and skin resistance) to the stressful interaction.

In the second study, Keane and Lisman (1980) tested a group of normal non-shy men using a similar design and an identical experimental procedure. Three dose levels were used: placebo, 0.26 g/kg and 0.69 g/kg. On the behavioural measures, the results replicated the findings of the first study and revealed a dose related impairment of social performance. Subjective anxiety was not affected by either alcohol content or instructional set. The ratings that each subject gave of his own performance were higher in the placebo group. The high-dose group showed greater HR increase during the social interaction. Skin resistance level was not affected by either alcohol or instructional set.

Wilson et al (1980) tested a group of male drinkers, ranging from light to heavy drinkers, in a tension-inducing social interaction similar to the one used in previous experiments, in which subjects had to interact with a female confederate after drinking either a placebo or 0.5 g/kg or

1.0 g/kg. Alcohol influenced HR increase during the stressing social interaction. This effect was dose dependent: the higher the dose, the smaller the increase in HR (the effect was statistically significant only for the high dose). Alcohol did not affect skin conductance or self-reported anxiety. There was no relationship between subject's drinking patterns and any of the measures of stress response.

Lipscomb et al (1980) used the same anxiety-inducing social task in which subjects, who were classified as high and low in tolerance on the basis of the effects of alcohol on body sway, drank one of two doses: 0.5 g/kg or 1.0 g/kg. HR increase was larger in the low-dose group than in the high-dose group. HR in the high-tolerance group increased more than in the low-tolerance group. Skin conductance changes did not discriminate between the doses. Both for HR and for SC changes there was a significant dose x tolerance interaction: "high-tolerance subjects were more aroused than were low-tolerance subjects at the small but not at the large dose, suggesting that high-tolerance subjects must consume more alcohol to achieve the same autonomic effect experienced by the low tolerance subjects" (p. 577).

Bradlyn et al (1981) used a BPD to test the effects of alcohol and expectancies in a situation of public speaking in a group of male social drinkers. The dose was 0.9 ml of vodka per lb of body weight. It was found an effect of alcohol content on the ratings of anxiety made by judges on the subjects' performances: the groups who drank a placebo were judged as more anxious. Alcohol did not influence changes of skin conductance. Expectancy did not have any influence, although it must be noted that the placebo manipulation was not successful in the group that was given alcohol and told that the drink did not contain

any alcohol.

Sher and Levenson (1982) reanalysed a previous experiment (Levenson et al, 1980, already mentioned), adding the analysis of the influence of individual differences in risk for alcoholism. It was found that subjects classified as high-risk for alcoholism on the basis of questionnaires showed a more pronounced alcohol-induced reduction in the increases of HR and subjective anxiety occurring during the stressful procedures (self-disclosuring speech or electric shock). In a second experiment reported in Sher and Levenson (1982) a confirmation of the role of individual differences was attempted. Two groups, high and low risk for alcoholism (classified by means of the Socialization scale of the Californian Psychological Inventory and the McAndrew Alcoholism Scale) and, who had identical current consumption rates of alcohol, were exposed to the speech stressing task. Half of the subjects in each group drank a placebo and the other half consumed 1.0 g/kg. Reduction of the stress response due alcohol was found to be smaller than in the first experiment. Confirming the mediating role of individual differences, alcohol reduced cardiac acceleration only in the high-risk group.

The role of individual differences was not confirmed in the experiment by Sher and Wallitzer (1986), in which subjects were allocated to one of three dose levels: placebo, 0.425 g/kg or 0.85 g/kg. The stressing task was again the self-disclosure speech. Alcohol reduced the HR increase during the stressing performance, and also, although to a lesser extent, the increase of self-reported anxiety. Risk for alcoholism did not influence the results. Expectancies of tension-reducing effects of alcohol ('Alcohol Expectancy Questionnaire') did not have any influence either.

Hull and coworkers (reported in Hull, 1987) found that alcohol reduced anxiety only in high-self-conscious individuals. Low and high-self-conscious subjects took part in a repeated measures experiment in which they were required to give speeches about themselves on two occasions. Both groups were sober in the first session and drank alcohol in the second session. Various psychophysiological measures (heart rate and skin conductance amongst them) were recorded. When sober high-self-conscious subjects showed greater physiological arousal than low-self-conscious individuals. Alcohol eliminated this difference.

Josephs and Steele (1990) have shown that the anxiolytic effects of alcohol depends on the concurrent attentional demands of a distracting activity. They found that anxiety during the period of time prior to giving an embarrassing speech was reduced by alcohol to the extent of the attentional demands imposed by a distracting activity. The more demanding the task was, the larger the relaxing effects of alcohol were. Josephs and Steele (1990) concluded that: "When one's attentional capacity is reduced by alcohol and the demands of ongoing activity, one has less attention for stressful thoughts, and, as a result, anxiety is reduced." (p. 123).

Social vs. nonsocial stressors

Marlatt's (1983) distinction between social and non social stressors in relation to alcohol effects, and his prediction that alcohol will antagonize the stress caused by social but not by nonsocial stressors, are not supported by these experiments. Positive and negative results have been found with both social and nonsocial stressors. Alcohol has

been found to reduce the stress caused by nonsocial stressors (Eisenhofer et al, 1986, Levenson et al, 1980, the two experiments reported in Lindman, 1983, Polivy et al, 1976); but a similar number of studies failed to prove any effect of alcohol (Dangerink and Fagan, 1978, Logue et al, 1978, Rimm et al, 1981, Sutker et al, 1982, Thyer and Curtis, 1981); and alcohol has even been found to increase stress (Dangerink and Fagan, 1978, Logue et al, 1978). Studies using social stressors provide a similar picture. A tranquillizing effect has been found in some studies (Wilson et al, 1980, Limpscomb et al, 1980, Sher and Levenson, 1982, Sher and Walitzer, 1986); although negative results have also been found (Wilson and Abrams, 1977, in both experiments reported in Keaman and Lisman, 1980); and Keaman and Lisman (1980) also found that alcohol increased the response to a social stressor.

Marlatt's hypothesis seems to be based on his idea that alcohol has no significant pharmacological effect on psychological functioning (see chapter 1), and that learned expectancies are the predominant factors of the effects of drinking. If alcohol has a real effect learned expectancies might interact with the pharmacological effects of alcohol, but they would not be as prepotent as Marlatt (1983) proposes.

Dose

The dose seems to be a more decisive factor than the nature of the stressor. The usage of a high dose (between 0.80 g/kg and 1.0 g/kg) appears to secure positive results (Eisenhofer et al, 1986, Lindman, 1983, Levenson et al, 1980, Wilson et al, 1980, Limpscomb et al, 1980, Sher and Levenson, 1982, Sher and Wallitzer, 1986); although exceptions to this rule are not totally absent. High doses (0.9 g/kg) have been

reported to increase tension (Dengerink and Fagan, 1978, Logue et al, 1978). Lower doses (0.4 - 0.5 g/kg) have also been found to reduce stress on some occasions (Polivy et al, 1976, Sher and Walitzer, 1986).

Dependent variables

Heart rate is the measure of stress that has provided the most consistent results. A high dose of ethanol (0.8 - 1.0 g/kg) has consistently been found to reduce cardiac acceleration to various stressors (Eisenhofer et al, 1986, Levenson et al, 1980, Sher and Levenson, 1982, Sher and Wallitzer, 1986, Wilson et al, 1980). In those studies in which a dampening of HR was not found, the failure may be explained by the low dose used (e.g. Wilson and Abrams, 1977).

Although some negative results have been reported, alcohol has been shown to reduce the subjective response of stress in a number of studies (Eddy, 1979, Lindman, 1983, Levenson et al, 1980, Polivy et al, 1976). Some of the negative results occurred in studies in which no tranquillizing effect of alcohol on HR was found (e.g. Keane and Lisman, 1980, Wilson and Abrams, 1977); although in some cases a reduction in HR was found, which was not reflected in the subjective measures (e.g. Wilson et al, 1980).

In those studies in which electrodermal activity has been recorded (usually, skin conductance level), the most frequent result has been that alcohol, whatever the level of dose used, did not influence this measure (Bradlyn et al, 1981, Levenson et al, 1980, Lindman, 1983, Sher and Levenson, 1982, Wilson et al, 1980).

4.7. Discussion

Survey studies seem to indicate that one important reason for drinking is to relieve tension and to cope with stress, although this is not the only thing that motivates people to consume alcohol.

It has been shown that stress can lead to higher consumption of alcohol. But also exposure to erotic stimuli has been shown to increase alcohol consumption (Gabel et al, 1980).

As Tucker et al (1982) concluded, the studies examining mood after drinking do not eliminate the possibility of tension-reducing effects of alcohol, although they cannot provide strong evidence in favour of the hypothesis.

Animal research in general is supportive of the tension reduction hypothesis.

The most relevant body of research consists of the studies examining the effects of alcohol on the response to a stressing situation. These studies as a whole indicate that a relatively high dose of alcohol (in the region of 0.8 g/kg to 1.0 g/kg) has an anxiolytic effect, which shows most consistently in the dampening of the HR increase provoked by the stressor.

The tranquillizing effect of ethanol has also been observed on self-reported stress or anxiety, although less consistently. The electrodermal response (change in skin conductance level has been the EDA index most frequently used) to a stressful situation does not seem to be

influenced by ethanol.

There are two possible explanations for the discrepancy between the effect of alcohol on the cardiac response and on the electrodermal activity. It may be that alcohol has a direct peripheral effect on cardiac response, possibly via beta-adrenergic blocking. However, alcohol has been found to dampen the stress-induced increase in plasma adrenaline, which suggests a central effect. Research on the effects of alcohol on the physiological response to stress in animals (Pohorecky, 1990) also supports the hypothesis of a central effect. Alternatively alcohol might have a central effect, which results in reduced cardiac acceleration but does not influence electrodermal activity. Sher (1987) has considered the possibility that alcohol affects the 'Behavioural Activation System' (BAS) but not the 'Behavioural Inhibition System' (BIS) of Gray's (1978) two-factor learning theory. According to Fowles (1980) EDA reflects BIS activation while HR reflects the activity of the BAS. Gray's model posits that the BIS underlies the inhibition of behaviour in punishment and is the substrate for anxiety, and that the BAS is an appetitive, approach system which responds to incentives. The explanation can be rejected on several grounds. The stressful stimuli or situations used in these experiments seem more likely to be related to the BIS. On the other hand, in animals alcohol has been shown to reduce anxiety in response to situations that Gray's model explicitly links to the BIS (Hodgson et al, 1979). Moreover, Gray's theory explicitly states that alcohol and anxiolytic prescription drugs will decrease the activity of the BIS.

Alternatively, it could be argued that the discrepancy between HR and EDA in these studies reflects the lack of reliability (or validity) of EDA

(and specifically skin conductance level) as an index of stress or anxiety. Sartory and Lader (1981) reviewed the psychophysiological measures of anxiety, mainly as used to distinguish between groups of anxious or phobic subjects and controls. They concluded that "heart rate is increased in anxiety states and fear, and in heightened fear states relates to the magnitude of experienced fear" (p. 172), but they noticed that "SCL is a fairly crude measure which will only separate extreme groups" (Sartory and Lader, 1981, p. 175). Similarly, Johnson and Lubin (1972) concluded that, while there is a demonstrated relationship between EDA and activation, "the efforts to correlate EDA with anxiety level have been unsuccessful or contradictory" (p. 404).

Amongst the factors determining the effects of drinking are the expectancies and beliefs about the effects of alcohol. It has been shown that the belief that alcohol has been consumed can reduce stress (e.g. Wilson and Abrams, 1977); but the opposite result has also been found (e.g. Abrams and Wilson, 1989, Dengerink and Fagan, 1978, Logue et al, 1978, Polivy et al, 1976); and a number of studies have failed to find any effect of expectancies (e.g. Bradlyn et al, 1981, Levenson et al, 1980). The investigation of the role of expectancies is limited by the difficulty in implementing the deception that the BPD entails at the dose range which has been shown effective in tension reduction (see Knight et al, 1986).

Expectancies and beliefs about the tension reducing effects of alcohol seem to be acquired by social learning before actual experience, as young inexperienced adolescents seem to have already precise ideas about what to expect from drinking. Arguably, actual experience of the effects of alcohol will later influence and shape these expectancies.

Moreover, folk wisdom about the effects of alcohol is likely to reflect to some extent the real effects of ethanol.

Both alcohol and the belief of having drunk alcohol have been found to increase stress on some occasions. One explanation of this could be that those subjects who have drunk or who believe that they have drunk alcohol become more anxious because they know that alcohol makes them less able to cope with a demanding task.

Steele (Steele et al, 1986, Josephs and Steele, 1990) has proposed a model in which alcohol does not affect anxiety directly, but its anxiolytic effects derive from its action on cognitive capacities. Josephs and Steele (1990) have shown that the reduction in anticipatory anxiety after alcohol consumption depends on the presence of a distracting activity. Steele suggests that, because the capacity for processing that requires attention is lowered by alcohol, if a simple distracting activity is provided the intoxicated subject will devote all his diminished attentional resources to the distracting task, and this will result in a reduction in anxiety. Steele's model can account for the reduction of anticipatory anxiety, but cannot explain the reduction of anxiety during the performance of a stressing task (such as the disclosure speech) as no distracting activity is present.

Hull (1981, 1987) also holds the view that alcohol affects anxiety only via the impairment of 'high-order cognitive processes'. According to Hull, the anxiolytic effects of alcohol are secondary to a reduction in self-awarenes, which in Hull's view depends upon high-order processing of self-relevant information. Hull (in the study reviewed above) showed that alcohol reduced physiological anxiety to a larger

extent in high-self-conscious subjects. However, as Hull himself recognizes, these results are not conclusive, as "it is possible that alcohol had its effects in this study by virtue of directly reducing physiologic responsivity, as opposed to indirectly reducing physiologic arousal by virtue of its effects on self-concious cognition" (Hull, 1987, p. 285). On the other hand, if alcohol reduces self-awareness this does not mean, as Hull assumes, that this effect is the result of impairment in higher-order cognitive processing. Bargh (1982) has demonstrated that self-relevant information is processed automatically. "On a dichotic listening task in which subjects attended to or ignored self-relevant stimuli, it was found that self-relevant information required less attentional resources when presented to the attended channel, but more when presented to the rejected channel, relative to neutral words." (Bargh, 1982, p. 425)

Chapter 5.

EFFECTS OF ALCOHOL ON SOCIAL ANXIETY: A REPEATED MEASURES EXPERIMENT.

5.1. Introduction

The review of the literature reveals evidence of a reduced stress response after the consumption of relatively high doses of alcohol, particularly if the cardiac response is used as measure of the response of the organism to the stressor. These anxiolytic effects of alcohol seem to be dose related. The effects of ethanol on cardiac response appear robust after the administration of doses between 0.8 and 1.0 g/kg. Although Sher and Wallitzer (1986) found a significant anxiolytic effect with a dose of 0.45 g/kg, doses below the level of 0.8 g/kg have usually failed to influence the stress response significantly.

Researchers investigating the effects of alcohol on various domains have pointed out that, because the action of alcohol presents great variability between individuals, a very strong effect of the drug is needed to reach statistically significant results, when between subjects designs are used. Therefore, the use of repeated measures designs has been advocated, in which each subject serves as his own control, to minimize the error variance and to make it easier to isolate the effects of the alcohol dose (Huntley, 1973; Porjesz and Begleiter, 1985; Maylor, Rabbitt and Kingstone, 1987).

The experimental technique of within subject comparisons has an egregious origin, as it was first described by the French physiologist Claude Bernard (1865), who called it 'method of successive differences'.

Since then repeated measures or cross-over designs have been extensively used, particularly in clinical and pharmacological trials. Despite the problems of this type of design (e.g. carry-over effects, drop-outs, etc.), of which researchers have become increasingly aware (Vere, 1979, Hills and Armitage, 1979), repeated measures designs are a useful method of minimizing variance.

The main objective of the present experiment was to test the possibility that a repeated measures design serves to demonstrate an effect on social stress of a low dose of alcohol. It has been claimed that the stress response caused by the speech anxiety condition in the self-disclosure task does not habituate (Abrams, 1983). If this proves to be true, the repeated measures design will be the technique of choice to investigate the effects of ethanol on social anxiety.

In the self-disclosure task a confederate to whom the subject has to talk has usually been used. The presence of the confederate (a person unknown to the subject and usually of the opposite sex) is a stressing factor itself, and by imposing on the subject the need to interact with this person it is assured that the stressing circumstance and the pressure on the subject to talk are kept for a sufficient period of time. In the present study the unavailability of a confederate was overcome by the introduction of a video camera to which the subject was required to address his speech. Additionally, the speaking period was divided into three phases with a new topic in each one. This was meant to maintain the stress for a longer period.

5.2. Method

5.2.1. Subjects

The subjects were 18 male social drinkers with an average age of 20.05 years, ranging from 18 to 24 (SD = 1.69). Recruitment was carried out mainly by advertisements placed at different locations around Edinburgh University campus. The subjects reported a customary drinking of 20.75 units of alcohol per week (SD = 10.36). Exclusion criteria were: being a teetotaller, a history of psychiatric illness or drinking problems and any condition incompatible with the intake of a moderate dose of alcohol (e.g. liver and kidney problems). Subjects took part in the study after signing an informed consent form (see Appendix). They were asked to fast for four hours, and not to drink alcohol or take any drug or medicine for 12 hours, before the time of the experimental sessions. Subjects were paid five pounds for their participation in the study, which included two experimental sessions, one or two weeks apart.

5.2.2. Design

A cross-over design was used for the reasons outlined above. All subjects participated in two experimental sessions. A placebo was administered in one of them and an alcoholic drink in the other. Half of the subjects had placebo on the first occasion and the alcoholic beverage on the second, whereas the other half went through the two experimental conditions in the opposite order. After the absorption period the subjects were subjected to the 'social stress task'. Subjective

and cardiac responses were measured during the task.

5.2.3. Stimuli, measures and apparatus.

5.2.3.1. Social stress task

Subjects were required to talk to a video camera for three minutes. The speeches were recorded in video tapes. The subjects were given three cards, each one containing a question or topic about which they had to talk. They were told that they had to try to present themselves as honestly as possible. They were also told that two judges were going to rate their performance and they were encouraged to try to give a good impression. Three pairs of cards had been prepared. Both cards in each pair referred to roughly the same topic. The contents of the six cards were:

1A.- Are you an attractive person?. Why?

1B.- Why should a boyfriend/girlfriend be interested in you?

2A.- Which family members do you like best?. Why?

2B.- Which family members do you like least?. Why?

3A.- What important life decisions will you make in the next five years? What will you be doing then?

3B.- Since leaving school, what have you done that you are proud of and disappointed about?

In each session subjects were asked to talk about the question posed by a card from each pair. In the first session, a card from each pair was drawn, and the selected cards were presented at a random order. On the second day, the remaining three cards were used after being shuffled.

5.2.3.2. Questionnaires and Visual Analogue Scales

Prior to the start of the first session, subjects were required to complete the Trait Form of the State-Trait Anxiety Inventory, STAI, (Spielberger et al 1970).

The "Mood Adjective Check List" (MACL, Mackay et al, 1978) was used to monitor changes in mood (arousal and stress). The subjects completed the MACL three times during the experimental session: prior to the administration of the drink, 20 minutes after the consumption of the beverage and after the social stress task. This instrument consists of two scales, arousal and stress. It assumes a two-dimensional model of mood, one dimension, stress, relating to hedonic tone, "general sense of well-being" or "feelings of unpleasantness/pleasantness", and the other, arousal, referring to "wakefulness/drowsiness or vigour" (Mackay et al, 1978; Cox and Mackay, 1985). The 30-adjective refined version was used in this study (see Cox and Mackay, 1985). A symmetrical answer format was preferred, where the subject was required to choose one of the following options: "I definitely do not feel ...", "I do not feel", "I feel slightly" and "I definitely feel" (see Appendix).

Bipolar Visual Analogue Scales (VAS) were used to measure subjective sensation of tension or anxiety and perceived alcoholic intoxication. The VASs consisted of 10 cm horizontal lines anchored on left and right sides by the terms 'totally tense' and 'totally relaxed' or 'totally sober' and 'totally drunk' (see Appendix).

5.2.3.3. Psychophysiological recording

Electrocardiogram (ECG) was recorded by means of an optically isolated 'Philip Harris ECG transducer', which was connected to the Analogue Port of a BBC-B Econet microcomputer through an optical fibre. The active electrodes consisted of disposable stainless steel electrodes placed on the forearm. The reference electrode was a silver impregnated "Velcro" tape, which was placed around the right leg, a few centimetres above the ankle. Hypertonic gel was used between electrodes and skin. Prior to attaching the electrodes the skin was rubbed with some cotton wool impregnated in alcohol.

The output from the ECG amplifier was fed into the built-in analogue-to-digital converter (ADC) of the BBC-B microcomputer. The converter was read every 20 ms (50 Hz). Each reading of the ADC was stored in form of 8-bit words in consecutive memory addresses through indirection operators. After every minute of physiological recording, the data accumulated in the computer memory were saved as memory blocks in a floppy disk. This operation took less than two seconds. This procedure allowed for continuous physiological recording with only 2-sec non-operational periods every minute.

5.2.3.4. Dose and placebo manipulations

Subjects were induced to believe that they had received alcohol on both experimental sessions. They were told that the aim of the experiment was to test the effects of two different doses of alcohol. In the 'alcohol' session subjects were given a dosage of alcohol aimed to produce a maximum BAC of 70%. The actual dose each individual had was calculated on the basis of his total body water and blood water using the equation given by Watson et al 1981). Total body water was calculated from anthropometric data using nomographs based on regression equations derived from empirical data (Watson et al, 1980). Ninety per cent pure alcohol was diluted into a mix of bitter lemon and lime juice, at the ratio 1:9:1 (alcohol:bitter lemon: lime juice). On the 'placebo' session, the subject had the soft part of the mix.

5.2.4. Procedure

On arrival the subject signed the consent form and completed the MACL and the Trait Anxiety STAI. The subject was then given the drink which might or might not contain alcohol. In either case the consumption time was 15 minutes. In order to keep the rate of consumption constant across subjects and sessions, the total dose was divided into five equal parts poured in five different glasses and the subject instructed to consume each subdose within three minutes.

Fifteen minutes after they finished the drink, the MACL was completed again. At this point the subject was escorted to the experimental

chamber. The subject sat down on an easy chair in front of the video camera. The equipment for psychophysiological recording was to the right of the subject. A curtain separated the subject form the recording instruments and the investigator.

After the electrodes had been attached, 25 minutes after finishing the consumption of the beverage, the experimenter gave the subject instructions to relax until the commencement of the social stress task. HR was recorded during the last minute of this 3-min relaxation period, which was followed by the social stress task, which consisted of the following phases:

Anticipation: At the end of the relaxation period the experimenter warned the subject that he would have to start talking after a minute.

Speech 1, 2 & 3: A minute after the warning the experimenter tendered the first card and informed: "This is the subject you have to talk about. Look at the camera all the time and do not stop talking." A minute later a second card was offered repeating the same instructions. The same for the third card.

Recovery: After the third speech the subject received instructions to relax. HR recording continued for the first minute of this relaxation period.

After the conclusion of the social stress task the subject reported how he had felt during each speech period on the VASs. Finally he reported how intoxicated he was feeling and how much alcohol he thought he had consumed.

After the second experimental session the subject was debriefed about the nature of the experiment and the content of the drinks he had really consumed.

5.2.5. Response quantification of psychophysiological data.

The average HR in each phase of the social stress task was calculated by means of a computer-helped procedure. Each 5-sec portion of ECG trace was shown on screen and the operator moved a line by pressing a key until it was placed on the right most QRS peak. Another line was placed on the left most peak and the operator entered the number of peaks in between. The program then returned the HR in beats per minute on screen. The resolution of this procedure is determined by the sampling rate, so that time lapsed between the two extreme most QRS peaks in every five seconds epoch was computed with a precision of +/- 20 ms.

5.2.6. Statistical analysis

The experiment was analyzed as a 2 x 2 repeated measures factorial design with the order in which the subjects went through the two experimental conditions as a grouping factor and the experimental treatment (alcohol or placebo) as a within subject factor. The BMDP P4V program (Dixon, 1985) for univariate and multivariate ANOVA was used for the analysis. This design can also be understood as a latin square with a grouping factor with two levels (the two groups receiving the treatments in different order), with treatment (alcohol and

placebo) as a within subject factor, and session (first or second session) as a within subject latin square factor (Winer, 1971). Both techniques are equivalent and yield identical results. The grouping and the treatment factors are equivalent in both schemes; the session or practice factor of the latin square solution is represented in the 2 x 2 ANOVA by the interaction term. In both schemes the grouping factor represents the interaction between treatment and session (see Winer, 1971, p. 716).

The Greenhouse-Geisser (1959) correction to the degrees of freedom was applied when the repeated measures factor had more than two levels in order to correct for the effects of lack of sphericity of the covariance matrix.

5.3. Results

5.3.1. Dose

The subjects received an average dose of 0.642 (sd=0.037) g/k (grams of ethanol per kilogram of body weight).

5.3.2. Trait anxiety

The group of 18 subjects as a whole had a mean score on the trait anxiety questionnaire of 37.16 (sd=5.96). The two order groups did not differ in this measure (p= 0.26).

5.3.3. Placebo manipulation checks

Subjects' estimates of the amount of alcohol consumed, given at the end of each session, are presented in table 5.1. and figure 5.1. A 2 x 2 (order group x dose) ANOVA revealed that dose - that is, whether the drink contained alcohol or not - did not influence the estimates ($F(1,16)=0.61$; $p=0.44$). However, the order factor and the interaction term produced statistically significant effects (respectively, $F(1,16)=4.83$; $p=0.043$ and $F(1,16)=49.15$; $p<0.0001$).

Data of ratings of perceived intoxication are given in table 5.2. and fig. 5.2. A 2 x 2 x 2 (order group x dose x phase) ANOVA was used to analyze these data. The dose influenced the perception of intoxication: $F(1,16)=32.71$; $p<0.0001$. Neither order group nor the 'dose x order group' interaction factor affected this measure. Phase (after absorption/after task) did not influence the sensation of intoxication, although the 'dose x phase' interaction was statistically significant ($F=6.05$, $df=1/16$, $p=0.0256$).

5.3.4. Psychophysiological measures

HR was averaged for each 1-min period. A 2 x 2 ANOVA (program BMDP 4V, Dixon, 1985) with a factor (order) varying between subjects and another (dose) within subjects, was performed on the mean heart rate in the 1-min relaxation period prior to the onset of the stressing task. This analysis revealed an arousing effect of alcohol consumption on HR ($F(1,16)=19.84$, $p=0.0004$).

In the analysis of the psychophysiological response during the situation of speech anxiety, differential scores with respect to initial levels taken in the period of relaxation were used. As alcohol influenced the initial resting cardiac response, absolute values of HR reflect both the effect of alcohol and that due to the stressing condition. Therefore, change scores are a better index of the response to the stressing task. Table 5.4. shows the product-moment correlation coefficients between initial levels and differential scores. Correlation coefficients were always low and statistically nonsignificant, indicating that cardiac response to the social stress task was independent of initial levels.

Mean values of HR changes during the social stress task are given in table 5.5. These data were subjected to a 2 x 2 x 5 ANOVA (order group x dose x phase in social stress task). There was no effect of the grouping variable ($F<1$) nor of the dose x group interaction ($F<1$). The phase effect was statistically highly significant ($F=37.71$, $df=1.58/25.32$, $p< 0.0001$). When alcohol was consumed HR increases tended to be smaller, which was reflected in a statistical trend in the dose effect ($F=3.38$, $df=1/16$, $p=0.0845$). On examining table 5.5. and fig. 5.3. it can be observed that alcohol markedly reduced HR during the three speech periods when it was consumed in the second session, but it seems that the alcohol effect was balanced out by a habituation effect (i.e. reduced response on the second occasion). The cardiac reaction during the anticipation and recovery periods followed quite a different pattern: the HR rise tended to be lower on the first session specially if alcohol was consumed on this first occasion. 2 x 2 (dose x order group) ANOVAS were performed on each phase separately. During the anticipation and recovery periods the session effect (i.e. the interaction term) resulted in

Table 5.1. Estimates of consumed alcohol in terms of pints of beer. Means and standard deviations.

group P - A		group A - P	
Alcohol	**Placebo**	**Alcohol**	**Placebo**
3.44 (1.21)	1.5 (0.43)	1.11 (0.6)	2.66 (0.61)
time 2	**time 1**	**time 1**	**time 2**

Table 5.2. Means and standard deviations of perceived intoxication ratings (100 mm VAS).

	group P - A		group A - P	
	Alcohol	**Placebo**	**Alcohol**	**Placebo**
after absorption	25.33 (13.75)	14.66 (10.07)	37.33 (24.30)	14.77 (9.66)
after task	30.55 (16.92)	12.66 (7.82)	42.11 (13.29)	11.22 (9.88)
	time 2	**time 1**	**time 1**	**time 2**

Fig. 5.1. Estimates of consumed alcohol given at the end of the experimental session.

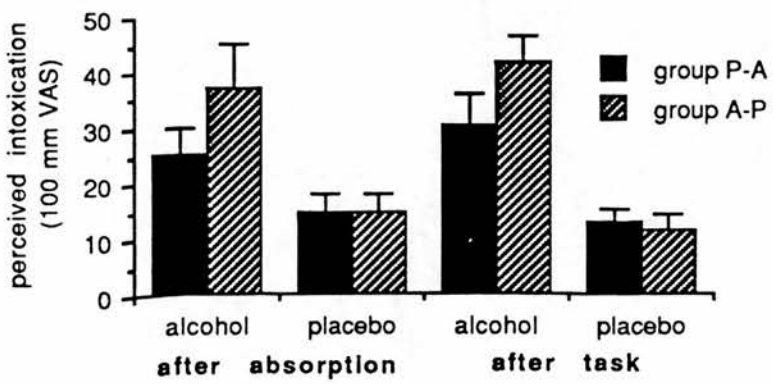

Fig 5.2. Perceived intoxication.

Table 5.3. Initial level of HR before the social stress task. Means and SDs.

	group P - A		group A - P	
	Alcohol	**Placebo**	**Alcohol**	**Placebo**
Heart Rate (bpm)	80.01 (16.82)	71.59 (13.17)	75.39 (11.02)	71.42 (12.14)
	time 2	**time 1**	**time 1**	**time 2**

Table 5.4. Pearson correlations between initial levels and change scores of heart rate during the social stress task.

	Placebo		Alcohol	
	r	**p**	**r**	**p**
anticipation	0.09	n.s.	-0.22	n.s.
speech 1	0.24	n.s.	0.07	n.s.
speech 2	0.34	n.s.	0.21	n.s.
speech 3	0.23	n.s.	0.02	n.s.
recovery	0.18	n.s.	0.21	n.s.

Table 5.5. Change of HR (bpm) in the social stress task. Means and standard deviations.

	group P - A		group A - P	
	Alcohol	**Placebo**	**Alcohol**	**Placebo**
anticipation	5.25 (6.72)	4.23 (5.78)	1.91 (4.56)	6.94 (4.74)
speech 1	22.89 (9.60)	26.24 (8.65)	22.27 (13.07)	23.05 (12.30)
speech 2	19.21 (13.15)	23.54 (14.26)	19.20 (12.68)	19.35 (9.96)
speech 3	14.06 (11.36)	21.74 (11.61)	15.55 (8.00)	15.25 (7.09)
recovery	4.32 (6.11)	2.16 (7.48)	1.50 (5.26)	4.34 (2.85)
	time 2	**time 1**	**time 1**	**time 2**

Table 5.6. Subjective feelings of tension-relax (100 mm VAS) in the social stress task.

	group P - A		group A - P	
	Alcohol	**Placebo**	**Alcohol**	**Placebo**
speech 1	45.11 (21.67)	24.00 (17.71)	37.55 (15.33)	51.88 (24.19)
speech 2	44.33 (19.33)	32.22 (21.74)	38.88 (19.56)	46.33 (20.60)
speech 3	57.77 (14.90)	36.77 (18.93)	50.33 (10.63)	61.00 (18.17)
	time 2	**time 1**	**time 1**	**time 2**

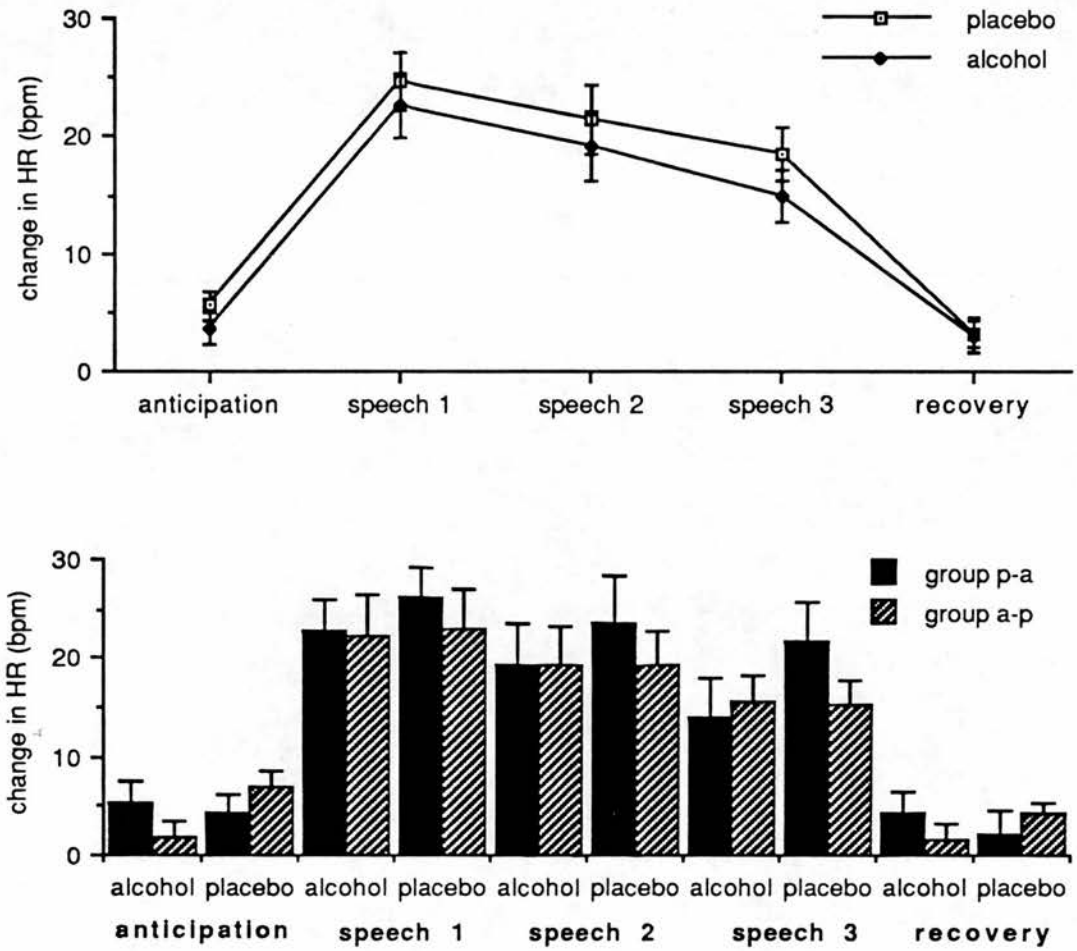

Fig 5.3. Changes in HR in the social stress task.

Table 5.7. Subjective arousal (MACL) during the experimental session. Means, standard deviation (in brackets) and adjusted means with the score before drinking as covariate.

	group P - A		group A - P	
	Alcohol	**Placebo**	**Alcohol**	**Placebo**
before drinking	32.3 (5.4)	28.8 (6.8)	30.6 (4.5)	32.3 (7.7)
after drinking	29.0 (6.9) *28.4*	30.5 (4.9) *31.6*	32.5 (5.3) *32.7*	28.4 (6.5) *27.8*
after task	30.5 (6.4) *29.9*	32.2 (5.2) *33.3*	31.2 (6.4) *31.4*	32.4 (7.1) *31.4*
	time 2	**time 1**	**time 1**	**time 2**

Table 5.8. Subjective stress (MACL) during the experimental session. Means, standard deviation (in brackets) and adjusted means with the score before drinking as covariate.

	group P - A		group A - P	
	Alcohol	**Placebo**	**Alcohol**	**Placebo**
before drinking	33.4 (3.3)	37.2 (2.2)	39.2 (4.7)	35.1 (4.0)
after drinking	31.8 (4.2) *33.6*	35.4 (3.0) *34.8*	36.4 (4.2) *34.5*	35.5 (4.0) *36.3*
after task	33.7 (5.1) *35.5*	40.5 (5.3) *39.9*	39.8 (4.8) *37.8*	34.4 (6.5) *35.2*
	time 2	**time 1**	**time 1**	**time 2**

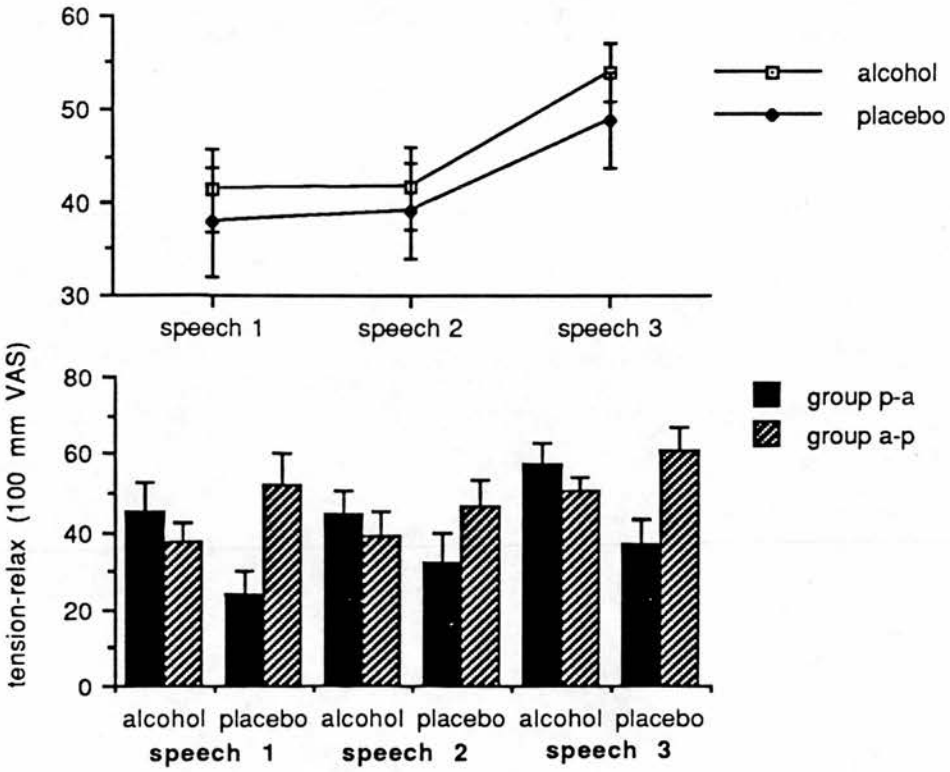

Fig 5.4. Subjective feelings of tension-relax during the social stress task.

statistical trends (F=3.08, df=1/16, p=0.098 for the anticipation phase; F=4.26, df= 1/16, p= 0.0556 for the recovery period). No effect of the main factors or the interaction was found during the first speech. There seems to be some influence of both dose and session in the second speech period, but the statistical tests were nonsignificant (dose effect: F=2.83, df=1/16, p=0.1118; 'dose x order group' interaction: F=2.47, df=1/16, p=0.1354). Both dose and session effects were clear in the third speech period (respectively F=5.83, df=1/16, p=0.0281 and F=6.82, df=1/16, p=0.0189). A 2 x 2 x 3 (dose x order group x phase) ANOVA on HR changes during the three speech periods revealed a marginally significant effect of alcohol (F=4.46, df=1/16, p=0.0508) and a session effect (i.e. 'dose x order group' interaction) also close to statistical significance (F=3.78, df=1/16, p=0.0697).

5.3.5. Subjective response

The data of subjective feelings of tension-relax that subjects reported on visual analogue scales are presented in table 5.6. and figure 4. A 2 x 2 x 3 (order x dose x phase) ANOVA revealed that alcohol did not influence the feelings of tension-relax (F(1,16)=0.69, p=0.418), although the order factor did (F(1,16)=4.87, p=0.042). The dose x order interaction was highly significant (F(1,16)=10.92, p=0.0045), reflecting a marked reduction in tension from the first to the second session. This session effect is clearly visualized in fig. 5.4. The factor phase also influenced significantly the sensation of tension-relax (F(1.90,30.47)=3.37, p=0.049).

2 x 2 x 2 (order group x dose x phase) ANCOVAs, with the scores before the administration of the drink as the covariate, were performed on the data from the arousal and stress scales of the MACL. The

BMDP P2V program (Dixon, 1985) was used for this analysis. Data from these variables are summarized in tables 5.7. and 5.8. The ANCOVA on stress measures revealed a significant linear relationship with the covariate at each group ($F=12.97$, $df=1/15$, $p=0.0026$) and at each dosage condition ($F=15.57$, $df=1/15$, $p=0.0013$). The group factor did not have any effect ($F<1$). Statistical trends were found both for the dose factor (interaction term $F=3.34$, $df=1/15$, $p=0.0875$) and the session factor ($F=3.33$, $df=1/15$, $p=0.0882$). There was a significant difference between phases ($F=6.09$, $df=1/16$, $p=0.0253$).

The ANCOVA on arousal data showed a significant relationship with the covariate at each group ($F=18.75$, $df=1/15$, $p=0.0006$) but a weaker relationship at each dose level ($F=3.68$, $df=1/15$, $p=0.0742$). Neither the group factor nor the alcohol influenced the feelings of arousal ($F<1$ in both cases). The session effect reached the level of statistical trend ($F=3.44$, $df=1/15$, $p=0.0836$).

5.4. Discussion

The social stress task succeeded in provoking a state of anxiety, which was reflected in a rise in HR during the anticipation phase of 5.58 bpm (sd=5.31) when placebo was consumed and 3.58 bpm (sd=5.83) when alcohol was administered. The HR rise peaked during the first speech period: 24.63 bpm (sd=10.44) with placebo and 22.58 bpm (sd=11.13) with alcohol. The anxiety response was also reflected in increased subjective stress.

The stressing effect of the speech anxiety task habituated from the first to the second session, producing a consistent statistical trend of the

session factor on the cardiac response. The habituation was stronger on the subjective measure of tension. Abrams' (1983) claim that the self-disclosure task did not habituate is strongly challenged by these results. In this experiment a confederate with whom the subject had to interact was not used. It is possible that by using a different confederate in each session the habituation effect would be reduced to a certain extent, but it seems unlikely that it would eliminate it totally.

Sher (1987) has hypothesized that the higher the level of anxiety the stronger the anxiolytic effect of alcohol would be. The results of this experiment do not support this hypothesis which has been formulated on exclusively empirical grounds, and based only on two experiments carried out in the same laboratory. Alcohol did not affect the cardiac stress response during the first speech period, when the response was highest, whereas its effects were apparent in the second and particularly in the third speech periods, in which the HR increase was less pronounced. It is impossible to conclude from this experiment whether this reduction was determined by a sequential factor (i.e. the alcohol might not affect a first reaction to the stressor but helps tranquillization after the initial burst of anxiety response) or by the level of the reaction (i.e. alcohol succeeds to reduce only lower levels of anxiety, but is ineffective in dampening stronger reactions).

These results provide some support for the hypothesis that alcohol has an anxiolytic effect at doses as low as 0.64 g/k. Although strictly speaking in statistical terms the effect of alcohol did not reach the conventional significance level of 5%, it should be kept in mind that ANOVA F-statistic represents a bidirectional test. As it can be argued that the hypothesis of this experiment (i.e. that a low dose of alcohol

reduces the stress response) was unidirectional, p-values from the ANOVA can be legitimately interpreted with some degree of liberality. The results of the present experiment provide no grounds to reject the thesis of the anxiety reducing effects of moderate or low doses of ethanol.

The estimates of consumed alcohol were not influenced by the actual intake of ethanol as reflected in the main effect of the dose factor. Subjects' estimates were largely determined by the session, with low estimates in the first session and higher estimates on the second. Dosage and session interacted. The inspection of table 5.1. and fig. 5.1. clearly shows that alcohol determined the estimates subjects gave on the second session, with those who drank alcohol giving higher estimates. More surprising is the tendency in the opposite direction that can be observed in the first session, with those who drank placebo estimating higher consumption of alcohol. It seems that subjects tried to guess on the basis of the information they were given. Having been told that they were receiving alcohol on both sessions, a low dose in one of them and a higher dose in the other, subjects in both groups seem to have thought that the dose received in the first session was not large enough and therefore concluded that they had been administered the low dose. Perceived intoxication followed a different pattern. The perception of drunkenness was strongly determined by the content of the drink. The session did not affect the perception of intoxication when placebo was administered, but subjects perceived themselves as more intoxicated when they drank the alcoholic beverage in the first session rather than in the second. When alcohol was actually consumed the perception of intoxication was higher after the speech anxiety task, whereas no change was observed through the

experimental session when placebo was administered. These results show that estimates of consumed alcohol and ratings of perceived intoxication are not at any rate equivalent.

It is worth noting the distinctive patterns in which the factors of the experimental design affected HR in the anticipation and recovery periods in contrast to the speech periods. The main determinant of HR in the anticipation and recovery periods was the session factor: HR rises were larger on the second session. For the anticipation period, a tentative explanation could be that the anticipation was more stressing after the subject had had real experience of stressing task. This, of course, does not explain the effect of session on the recovery period.

Alcohol did not affect self-reported arousal. Statistical trends which suggest effects of alcohol and session were observed on the stress scale. These effects were on the direction of the effects on HR during the speech periods.

In summary, this study has found that the self-disclosure task shows habituation, which eliminates the potential advantage of using a cross-over design. Despite this, this experiment provides limited support for the hypothesis that moderate (below 0.8 g/kg) doses of ethanol have anxiolytic effects.

Chapter 6.

EFFECTS OF ALCOHOL ON SOCIAL ANXIETY: A BETWEEN SUBJECTS EXPERIMENT

6.1. Introduction

When examining the effects of alcohol during a complex situation, like the 'social stress task' or the 'self-disclosure task', the interpretation of the observed physiological effects of alcohol is not straightforward. Alcohol might influence the primary response to the stimulus situation resulting in a dampened physiological reaction of stress; alternatively, the reduced HR increase might be the result of the influence of alcohol on processes occurring later in the 'emotional cycle'. Thus, alcohol might impair or distort the perception of the primary physiological reaction, and this would influence the secondary appraisal of the situation, affecting the subsequent physiological response. For example, intoxicated subjects might not perceive their cardiac response and, therefore, they would appraise the situation as less stressful, resulting in a reduced cardiac response. In chapter 3, some evidence was found suggesting that intoxicated subjects were less accurate in the perception of their physiological sexual response. Impairment in the scanning of both environmental and self information has been suggested as an important feature of the cognitive deficit in alcoholics (Robertson, 1985). The present experiment tested the possibility of a reduced perception of cardiac response in intoxicated subjects by examining the relationship between cardiac response and subjective perception of cardiac acceleration during the Social Stress Task (SST).

In the studies examining the effects of alcohol on stress, the dampening

of the cardiac response has not been accompanied by an effect of alcohol on electrodermal activity (see chapter 4). The measure of EDA that most studies have used has been the skin conductance level. Sartory and Lader (1981) reviewed the use of psychophysiological measures in states of anxiety and phobia in normal controls and clinical groups and concluded that 'SCL is a fairly crude measure which will only separate extreme groups, i.e. severe phobics and controls,' whereas 'spontaneous fluctuations (...) have so far yielded the most consistent results when investigating anxiety states in severely phobic patients and monosymptomatic volunteers alike' (p. 175). The present experiment examined the effects of alcohol on these two parameters of EDA in response to social stress.

A dose of 0.8 g/kg was used. This dose is within the range that has been shown to reduce the stress response, particularly as indexed by cardiac acceleration.

6.2. Method

6.2.1. Subjects

The subjects of this experiment were 22 male students with an average age of 21.09 years (sd= 3.006) and a mean weekly customary drinking of 20.89 (SD=12.74). Half of them received an alcoholic beverage while the other half were given a placebo. Both groups were told that they were to receive an alcoholic drink. Exclusion criteria were the same as in previous experiments. Prior to taking part in the study each subject gave signed consent. Three pounds were awarded to each volunteer for his collaboration. Subjects were required not to take any medicine, drug

or alcohol for a period of 12 hours, and not to eat for four hours, prior to the experimental session.

6.2.2. Design

Each subject was allocated to one of the experimental groups: placebo or alcohol. The experimental design also controlled for the order of presentation of the three topics the subjects addressed in the social stress task. After the allocation to one of the experimental groups, each subject was randomly assigned to one of the six permutations of the three topics. After the absorption period subjects engaged in the Social Stress Task (SST) ([1]). The dependent variables were heart rate (HR), electrodermal activity (EDA), subjective feelings of anxiety, and self-reported arousal and stress. Perceived intoxication and estimates of the amount of alcohol consumed were also recorded.

6.2.3. Stimuli, measures and apparatus

6.2.3.1. Social Stress Task (SST)

The SST was similar to the one used in the previous experiment (Chapter 5). As each subject participated only in one session, only three topics were used:

(a) Are you an attractive person? Why?

[1]Before this, the subjects had been subjected to a procedure to examine the startle and defence responses, which will be reported separately in the next chapter.

(b) Which family members do you like least? Why?

(c) Since leaving school, what have you done that you are proud of or disappointed about?

The instructions given to the subject were identical to the ones used in the previous experiment (see chapter 5).

6.2.3.2. Questionnaires and Visual Analogue Scales

Subjects completed the 'Mood Adjective Check List' (MacKay et al, 1978) three times during the experimental session. Subjective feeling of tension or anxiety was measured by means of bipolar visual analogue scales (VAS) consisting in 100-mm horizontal lines with the terms 'calm, relax' to the left and 'tense, anxious' to the right. Similar scales with the terms 'totally sober' and 'totally drunk' anchored to each side were used to measure perceived feelings of alcohol intoxication. Bipolar VASs were also used to measure sensations of cardiac acceleration ('heart beating normally' - 'heart beating very fast').

The Trait part of the STAI (Spielberger et al, 1972) was completed before the administration of the drink.

6.2.3.3. Psychophysiological variables

As in the previous experiment, electrocardiac activity was recorded by means of an 'ECG Phillip Harris' amplifier connected to the analogue port of an 'Econet BBC-B' microcomputer through an optic fibre. In order to avoid movement artifacts and to obtain a recording suitable

for automatic analysis, electrodes were placed on the thorax: the reference electrode, below the right breast, and the two active electrodes, one on the sternum and the other below the left breast (Brener, 1980). Reversible Ag/AgCl electrodes were used. The contact with the skin was through a hypertonic gel. Prior to attaching the electrodes, the skin was rubbed with cotton wool impregnated in alcohol in order to reduce skin resistance.

Skin conductance (SC) was measured by means of an active circuit device of the type described by Lowry (1977). Optical isolation was attained by adding an extra step, consisting in an optically isolated amplifier set up for a nominal gain of one (see Appendix for circuit diagram and specifications). 'Grass' eight-millimetre diameter Ag/AgCl electrodes were attached in a bipolar setting to the medial phalanx of the index and middle fingers. This gave a total electrode-skin contact area of 1 cm2. The constant voltage across electrodes was 1.2 volt. Contact between electrodes and skin was through a paste which contained a 0.05 M NaCl solution, and used agar-agar as thickening agent, prepared according to instructions by Venables and Christie (1980).

A computer program was developed, in which the A/D converter was controlled by an assembly language routine, which allowed a sampling rate of 100 Hz on each channel utilized (see Appendix).

During the SST the recording of the psychophysiological variables was performed continuously for periods of 66.65 seconds with lapses of 5 seconds. For each period of 66.65 sec data were temporally stored in RAM memory, and then the accumulated memory block of 6656 bytes

transferred to a floppy disc. Five seconds were allowed for the transfer, after which the recording was resumed.

6.2.4. Dose and placebo manipulation

Subjects were told that the objective of the experiment was to compare the effects of two different doses of alcohol.

Subjects in the alcohol group received a dose of 0.8 g of ethanol per kg of body weight. As in previous experiments 90 % pure ethanol was mixed with bitter lemon and lime juice in a ratio of 1:9:1 (pure alcohol:bitter lemon:lime juice). The subjects allocated to the placebo group consumed only the non-alcoholic part of the mix.

6.2.5. Procedure

All experimental sessions took place between 16.00 and 20.00. After signing up the consent form (in which the mildly stressing nature of the experimental procedure was explained), the subject was weighed and then he completed the Trait STAI, MACL and VASs. The experimenter poured the drink in six equal glasses and instructed the subject to drink each glass in a maximum of three minutes. This was in order to keep the rate of consumption constant across subjects. The total consumption time was therefore 18 minutes. The drink contained alcohol for half of the subjects.

After consuming the beverage an absorption period of 25 min was allowed, during which the electrodes for ECG and EDA recording were attached. MACL and VASs were completed at the end of this period.

Between 30 and 40 minutes after the end of consumption, the test of startle and defense responses took place. It will be reported in the next chapter. After this, the subject completed the VASs again. The experimenter then gave the instructions for the social stress task (SST):

"You will be asked to talk to a video camera for three minutes. You will talk openly and honestly about yourself.

Your speech will be recorded and two people who do not know you will view it independently and rate it in several dimensions - attractiveness, fluency, honesty, etc.. Try to make a good impression.

You will talk about three different aspects of yourself. The experimenter will give you a card prompting an aspect or specific area of your life, and then you will talk about that topic for one minute. After that, you will be given a second card, and finally a third one.

Keep talking all the time! Do not hesitate. Do not stop until the experimenter gives you the next card or until he indicates you to finish the third speech.

If you think you do not have any thing to say about that particular topic, just explain why you think that for one minute. In any case, keep talking!

Remember, try to make a good impression."

The experimenter put the video camera in place in front of the subject and instructed him to relax for few minutes. The experimenter and the equipment for the recording of psychophysiological data remained behind the subject, out of his sight, during the experiment. After approximately five minutes of relaxation, during the last of which ECG and EDA were recorded, the experimenter instructed the subject to indicate how he felt on the VASs and warned him that after a minute he would have to start talking, while focusing the camera on him and switching it on. The anticipation period, which lasted 1 min, started at this moment. It was followed, as in the previous experiment (Chapter 5), by three speech periods and the recovery period in which instructions to relax were given. Each period lasted 1 min, with 10 seconds in between to complete the VASs.

At the end of the session the subject completed the MACL and the VASs and estimated the alcohol he had consumed. The subject was then given information of the real content of the drink.

6.2.6. Data reduction and response quantification

The ECG trace was analyzed off-line by means of a computer program (see Appendix) that performed algorithmic detection of QRS peaks,(1) measured inter-beat intervals and calculated HR second by second. Then, the average HR for each phase of the SST was calculated.

The program for off-line processing of the SC recording firstly calculated the SC value in micromhos for each stored data point (a sample every 10 milliseconds) as a function of the output voltage and

(1) See Appendix for definition of psychophysiological terms

the suppression voltage used at each moment. SC data were then plotted, using a dot-matrix printer, with a resolution of 4.2 cm per micromho and 3.5 mm per second. The scoring was done by hand. The average skin conductance level (SCL) and the number of non-specific responses (NSR) were measured for each SST phase. For each phase the SCL was measured at 20-sec intervals (0, +20, +40, +60) or closest point free of NSRs and then averaged. These measures were taken to the nearest millimetre, which after conversion to micromhos rendered a resolution of +/- 0.023 micromhos. Any increase in SC larger than 0.05 micromhos was considered a NSR. The number of NSR in each SST phase was counted.

6.3. Results

6.3.1. Trait Anxiety

Both groups had similar Trait Anxiety scores ('alcohol group': mean=40.00,sd=10.64; 'placebo group': mean=38.00, sd=10.16, t=0.43, df=20, p=0.66).

6.3.2. Subjective variables

VAS of anxiety. Group means and standard deviations of self-reports of anxiety are presented in table 6.1. Although there was no difference in Trait Anxiety between groups, the 'placebo group' reported higher anxiety at the beginning of the experimental session, immediately after the experimental procedure had been explained to the subject and he had signed the consent form, and before the drink had been served. The difference was statistically significant (t=3.62,df=20,p= 0.002). This

difference faded gradually (see table 6.1.) and 40 minutes after the consumption of the beverage the difference had disappeared totally (means: 24.00 vs. 24.54, p= 0.94). The difference returned after the detailed instructions for the SST were given (means: 26.40 vs. 42.63; p=0.078). It seems that the 'placebo group' found more threatening the description of the task ahead than the 'alcohol group' and that this was not related to the content of the drink as it happened also prior to the consumption. During the SST the feelings of anxiety increased in both groups. Taking the reports emitted just prior to the anticipation phase of the SST as the initial levels, the increments from this point were larger in the 'alcohol group'. By the end of the SST both groups showed similar levels of anxiety.

MACL: stress and arousal. Before drinking, the 'placebo group' also scored higher on the stress scale of the MACL (contrast based on 2 x 3 ANOVA: $F(1/18)=6.24$, $p=0.022$). The difference was somewhat mitigated after the absorption period (see table 6.3.): $F(1/18)=1.77$, $p=0.20$. At the end of the experimental session both groups presented similar scores of stress ($p=0.82$).

Data of arousal are given in table 6.2. Arousal slightly decreased after the absorption period. There was no difference between groups. The group effect in the 2 x 3 (group x phase) ANOVA produced an $F<1$.

6.3.3. Heart rate

Average HR in the different 1-min periods in which it was recorded during the experiment is given in table 6.4. The changes in HR during the SST, taking as initial level the mean HR during the last minute of

relaxation prior to the anticipation phase of the SST, are shown in table 6.5.

After the absorption period the HR in the alcohol group was higher, although the difference was not statistically significant (t=0.83, df=19, p=0.41). During the minute of relaxation prior to the SST the difference between groups had increased but still was not statistically significant (t=1.11, df=19, p=0.27). It is worth noting, however, that when robust tests were applied the differences became closer to statistical significance. After absorption: trimmed t=1.50, df=15, p=0.15; before SST: trimmed t=1.87, df= 15, p=0.081.

To analyze the cardiac response during the SST, differential scores with respect to the mean HR in the relaxation period prior to the onset of the SAT were used. These data are presented in table 6.5. Differential scores were not correlated to the initial levels (see table 6.6.).

A 2 x 5 (group x phase of the SST) ANOVA ([2]) was performed on the change scores of HR. In general the placebo group showed higher increases, which was reflected in a significant main group effect (F(1/19)=4.43, p=0.048). The phase effect was highly significant (F(2.05/38.89)=39.10, p<0.0001). The group x phase interaction was marginally significant after the Greenhouse-Geisser adjustment (F(2.05/38.89)=3.15, p=0.053). To explore the phase effect and the interaction further, a contrast comparing HR changes during the speech periods versus the anticipation and recovery periods was performed. This revealed that HR increments during the speech periods were

[2]All ANOVAs were performed by the program P4V of the BMDP statistical package (Dixon, 1985).

significantly larger than during the other two phases ($F(1/19)=49.24$, $p=0.0001$). A significant interaction of this contrast with group ($F(1/19)=49.24$, $p=0.0001$) reflects the higher differences between speech and non-speech phases in the placebo group ($F(1/19)=0.032$).

HR increments during the speech phases were associated to the pre-drinking self-reported anxiety (see table 6.7).

6.3.4. Electrodermal activity

Skin conductance level (SCL). Mean SCL data (log SCL) from each of the seven 1-min periods recorded in the experiment are presented in table 6.8. SCL during the two relaxation periods, after absorption and prior to the SST, was higher in the placebo group although the differences were not statistically significant (after absorption $t=1.53$, $df=20$, $p=0.14$; before SAT $t=1.85$, $df=20$, $p=0.079$). SCL in the relaxation period after absorption tended to be related to pre-drinking levels of anxiety ($r=0.41$, $df=20$, $p<0.10$).

Table 6.9. shows the changes in SCL (log SCL - log SCL) during the SST. These change measures did not correlate with the initial levels (see table 6.10.). A 2 x 5 (group x phase of SST) ANOVA was applied to these differential scores. A significant grand mean evinced that SCL increased significantly from initial level ($F(1/19)=10.10$, $p=0.005$). No effect of group was found ($F<1$). The phase effect was statistically significant ($F(1.78/36.86)=0.0014$). There was no group x phase interaction effect ($F<1$).

Table 6.1.- Self-reports of anxiety at different moments during the experimental session using 100-mm VAS. Means (and SD).

	alcohol	placebo
Before drinking (-18 min)	16.54 (8.52)	44.09 (23.78)
After absorption (+30 min)	12.36 (11.02)	21.27 (14.09)
Before Social Stress Task (+40 min)	24.00 (17.76)	24.54 (15.73)
Before anticipation	26.40 (16.48)	42.63 (22.62)
After anticipation	43.90 (25.11)	49.45 (21.98)
After speech 1	41.18 (23.51)	50.18 (24.00)
After speech 2	43.18 (24.51)	53.54 (21.70)
After speech 3	46.81 (24.96)	42.30 (19.90)
After recovery	32.45 (19.84)	31.40 (18.39)
End of experimental session (+55 min)	28.90 (26.27)	27.09 (22.72)

Table 6.2.- Means (and SDs) of self-reported stress (MACL)

	alcohol	placebo
Before drinking	33.81 (7.66)	41.36 (6.56)
After absorption (+25 min)	28.27 (6.85)	34.63 (8.51)
End of experimental session (+55 min)	37.36 (10.50)	38.33 (8.91)

Table 6.3.- Means (and SDs) of self-reported arousal (MACL).

	alcohol	placebo
Before drinking	30.72 (5.27)	31.81 (6.06)
After absorption (+25 min)	27.09 (5.75)	28.45 (4.98)
End of experimental session (+55 min)	30.63 (6.63)	32.55 (5.07)

Table 6.4.- Means and SD of HR (bpm) after absorption and during the SST. Averages of 1-min recording periods.

	alcohol	placebo
After absorption (+30 min)	70.65 (8.22)	67.1 (11.17)
Before anticipation	76.80 (7.21)	71.98 (12.21)
Anticipation	82.24 (7.37)	80.70 (15.42)
Speech 1	89.68 (9.91)	92.32 (18.27)
Speech 2	83.11 (7.84)	84.59 (18.57)
Speech 3	82.41 (7.51)	83.81 (17.56)
Recovery	77.15 (6.52)	70.16 (14.50)

Table 6.5.- Means (and SDs) of HR changes (bpm) during the SST.

	alcohol	placebo
Anticipation	5.18 (4.24)	8.43 (7.81)
Speech 1	12.89 (4.93)	20.36 (8.92)
Speech 2	6.32 (3.94)	12.63 (9.69)
Speech 3	5.62 (5.09)	11.85 (7.28)
Recovery	-0.36 (3.76)	-1.80 (4.82)

Table 6.6.- Pearson correlations between initial levels and change scores of HR during the SST (n=21).

	initial level	
	r	p
Anticipation	-.02	n.s.
Speech 1	.25	n.s.
Speech 2	.17	n.s.
Speech 3	.16	n.s.
Recovery	.11	n.s.

Table 6.7.- Pearson's correlations between baseline predrinking measures of subjective anxiety and cardiac response of SST.

	pre-drinking subjective anxiety	
	r	p
Anticipation	.13	n.s.
Speech 1	.435	<0.05
Speech 2	.33	n.s.
Speech 3	.437	<0.05
Recovery	-.006	n.s.

Table 6.8.- Average skin coductance level (log SCL). Group means (and SD).

	alcohol	placebo
After absorption (+30 min)	0.50 (0.26)	0.64 (0.15)
Before anticipation	0.49 (0.26)	0.65 (0.09)
Anticipation	0.51 (0.27)	0.65 (0.10)
Speech 1	0.55 (0.27)	0.68 (0.11)
Speech 2	0.55 (0.28)	0.69 (0.11)
Speech 3	0.54 (0.28)	0.69 (0.12)
Recovery	0.52 (0.28)	0.67 (0.12)

Table 6.9.- Changes of skin conductance level (log SC - log SC) during the SST. Means (and SDs).

	alcohol	placebo
Anticipation	0.014 (0.017)	-0.003 (0.012)
Speech 1	0.063 (0.054)	0.029 (0.030)
Speech 2	0.054 (0.067)	0.026 (0.043)
Speech 3	0.049 (0.070)	0.027 (0.048)
Recovery	0.029 (0.063)	0.010 (0.048)

Table 6.10.- Pearson correlations between initial levels and change scores of SCL during the SST (n=22).

	initial level	
	r	p
Anticipation	-.02	n.s.
Speech 1	-.03	n.s.
Speech 2	.01	n.s.
Speech 3	.03	n.s.
Recovery	.11	n.s.

Table 6.11.- Frequency of NSRs.

	alcohol	placebo
After absorption (+30 min)	1.36 (2.29)	1.45 (1.69)
Before anticipation	3.91 (4.06)	3.18 (3.25)
Anticipation	7.36 (4.15)	5.54 (3.20)
Speech 1	8.91 (5.70)	5.54 (4.46)
Speech 2	7.27 (5.19)	4.90 (3.69)
Speech 3	6.09 (4.06)	3.90 (3.11)
Recovery	2.91 (2.73)	2.30 (2.45)

Table 6.12.- Pearson's correlations between changes in frequency of NSR and the initial level during the SST (n=22).

	r	p
Anticipation	-.63	<0.05
Speech 1	-.46	<0.05
Speech 2	- .59	<0.01
Speech 3	-.65	<0.01
Recovery	-.60 (3.89)	<0.01 (3.19)

Table 6.13. Self-reports of sensations of 'heart beating fast', using 100-mm VASs. Means (and SD).

	alcohol	placebo
Before anticipation	27.90 (17.39)	31.72 (22.20)
After anticipation	40.36 (25.11)	40.45 (21.98)
After speech 1	40.00 (19.43)	42.09 (22.72)
After speech 2	39.00 (20.24)	42.90 (20.59)
After speech 3	41.63 (24.79)	44.00 (19.92)
After recovery	34.18 (21.33)	26.50 (21.98)

Table 6.14. Pearson's correlations between feelings of heart beating fast and (1) heart rate, and (2) HR changes (n=21).

	HR		HR changes	
	r	p	r	p
Relaxation	-.37	n.s.	---	---
Anticipation	-.10	n.s.	.12	n.s.
Speech 1	-.07	n.s.	.14	n.s.
Speech 2	-.02	n.s.	.22	n.s.
Speech 3	-.13	n.s.	.07	n.s.
Recovery	-.18	n.s.	-.10	n.s.

Table 6.15.- Self-reports of feelings of drunkenness on 100-mm VASs. Means (and SDs).

	alcohol	placebo
After absorption (+30 min)	47.45 (27.96)	11.36 (10.80)
Before Social Anxiety Task (+40 min)	46.09 (27.33)	4.72 (5.27)
End of experimental session (+55 min)	43.90 (25.23)	4.00 (5.15)

SCL during the speech periods was higher than during the other two phases ($F(1/19)=41.34$, $p<0.0001$), but this difference was similar in both groups (contrast x group interaction $F<1$, $p=0.55$).

Non Specific Responses (NSR). Frequencies of NSRs for each group on the different experimental phases are given in table 6.10. The number of NSRs increased from the end of the absorption period to the start of the SST. There was no difference between groups.

Changes in the occurrence of NSRs from the relaxation period prior to the SST were computed. Differential scores were significantly correlated with the initial levels (see table 6.11.). Therefore, a 2 x 2 (group x phase) ANOVA was performed on raw frequencies of NSRs. There was a significant effect of phase (after Greenhouse-Geisser correction to degrees of freedom $F(3.23/61.40)=16.71$, $p<0.0001$). Except for the period after absorption and the recovery period, the frequency of NSR tended to be larger in the alcohol group, but neither group effect nor the interaction were statistically significant (respectively $F(1/19)=1.17$, $p=0.29$ and $F(3.23/61.40)=1.11$, $p=0.354$).

6.3.5. Perception of heart beating

VAS reports of perception of 'heart beating fast' during the SST (see table 6.13) were analyzed by means of 2 x 6 (group x phase) ANOVA. Groups did not differ in their perceptions of cardiac acceleration ($F=0.42$, $df=1/17$, $p=0.52$). There was a significant variation through phases ($F(3.77/64.02)=5.95$, $p=0.0001$), but this variation was similar in both groups (interaction: $F<1$).

Perception of cardiac acceleration was not correlated with HR or HR change (table 6.14).

6.3.6. Checks of placebo manipulation

A 2 x 3 (group x phase) ANOVA revealed a highly significant group effect ($F(1/20)=23.80$, $p=0.0001$): the alcohol group consistently reported higher levels of 'drunkenness' throughout the experimental session. The phase effect approached statistical significance after Greenhouse-Geisser adjustment ($F(1.62/32.43)=3.27$, $p=0.06$). The feelings of intoxication tended to fall from the first report taken 30 min after consumption to the last one 25 min later. The interaction was not statistically significant ($F<1$).

While the alcohol group, at the end of the experimental session, estimated to have consumed the equivalent to 3.45 pints of beer, the placebo group estimated their consumption in 1.22 pints. This difference was statistically significant ($t=6.30$, $df=18$, $p<0.0001$).

6.4. Discussion

The baseline differences in subjective anxiety and stress cannot be explained by differences in trait anxiety. The fact that the difference between groups was present before drinking, faded, and then reappeared again after the instructions for the SST, suggests that the two groups did not react to the description of the task (given at the beginning of the session and just before the SST) in the same way. It is impossible to be certain of the cause of this difference. However, going

back to the actual running of the experiment, a flaw can be pointed out that may lie at the basis of the unexpected pre-drinking differences. Due to some delay in the arrival of the ethanol the first recruited subjects were directly allocated to the placebo group. Almost half of the placebo group (five subjects) had completed the experiment before any 'alcohol subject' had been run. Perhaps the way the experimenter conducted the experiment changed inadvertedly, so that the subjects were less threatened by the description of the SST in later stages in the running of the study. Unfortunately, it was not only the reaction to the description of the task that was affected, but also the stressing nature of the SST, as it is revealed in the correlations between baseline pre-drinking feelings of anxiety and cardiac responses during the SST. This renders the reduction in cardiac response during the speech phases very difficult if not impossible to interpret. No statistical technique can overcome this problem. Analysis of covariance is meant to be used only when the experimental groups do not differ in the pre-treatment covariate (Howell, 1989; Warner and Bancroft, 1986). Thus, although the HR results of this experiment do not contradict previous data, they cannot add any support to evidence hitherto accumulated.

The increase in resting HR caused by alcohol was smaller than expected. Higher levels of anxiety in the placebo group which might have caused some increase in HR may account for this. The slightly elevated SCL in the two relaxation periods can also be attributed to the increased level of anxiety in the placebo group.

Although they did not reach statistical significance it is worth noting the larger increases in the frequency of NSRs in the alcohol group. If these group differences represent a genuine if small effect of alcohol

this should be interpreted as an increase in arousal rather than anxiety. This interpretation is based on two facts. On one hand, NSRs mainly reflect level of arousal (see Lader, 1980); on the other hand, variations of the frequency of NSRs in this experiment were not associated with levels of subjective anxiety.

Although it can be affirmed that placebo manipulation was successful in that all subjects apparently believed they consumed alcohol, the results on perceived intoxication reveal, as in the previous experiments, that those who were administered a placebo did not feel as strong symptoms of intoxication as did the alcohol group.

Chapter 7.
EFFECTS OF ALCOHOL ON THE PSYCHOPHYSIOLOGICAL RESPONSE TO HIGH-INTENSITY AUDITORY STIMULI

7.1. Introduction

This chapter describes a study which examines the effects of alcohol intoxication on the primary response to stressing environmental stimuli. This study has been carried out within the paradigm and framework of the 'defense reflex model'.

Pavlov (1923) observed that his laboratory dogs failed to emit the conditioned response they had previously learned when a novel stimulus coincided with the presentation of the conditioned stimulus. Pavlov (1923) called the response to the novel stimulus that interfered with the conditioned response 'orienting' or 'what is it' reflex. Sokolov (1963) distinguished two systems of response to environmental stimuli: the orienting reflex (OR), which enhances sensitivity and facilitates sensory processing, and the defense reflex (DR), which is elicited by more intense or painful stimuli and reduces the sensitivity of the sensory analyzers. Sokolov worked within the tradition of Russian reflexology. The publication of his book 'Perception and the Conditioned Reflex' in English in 1963 stimulated in the West the study of the OR and DR and their phsychophysiological correlates. Sokolov (1963) described two patterns of peripheral vascular responding associated respectively to OR and DR. The psychophysiological OR was said to consist of digital vasoconstriction and forehead vasodilation, and to show fast habituation. The DR would consist of digital and cephalic vasoconstriction and it would show slow habituation.

Sokolov's conceptualization of OR and DR resembles Lacey's conception of the organismic reactions of 'intake' or 'rejection' of environmental stimuli. According to Lacey (Lacey and Lacey, 1970) the organism can react to stimulation either in a receptive way with sensory facilitation, or rejecting environmental stimuli with sensory inhibition. These two response modes would be associated to characteristic patterns of autonomic responding. Graham and Clifton (1966) established the link between Russian tradition and Western research and extended Sokolov's conceptualization of OR and DR to include Lacey's notion of 'directional fractionation', which describes HR changes associated to the different ways the organism can react to stimulation. Lacey (1972) demonstrated that stimulus rejection is associated with HR increase whereas readiness for stimulus intake is related to cardiac deceleration. Graham (Graham and Clifton, 1966) pointed to the correspondence between the OR-DR model and the 'intake-rejection hypothesis', and suggested that the OR would be associated with cardiac deceleration and DR with cardiac acceleration, which was confirmed by Raskin et al (1969). However, Raskin did not find the cephalic vasodilation characteristic of the OR. The subjects responded with vasoconstriction at the forehead to both low and high-intensity stimulation. A review of the literature led Graham (1979) to conclude that "the critical cephalic vasodilation response has proven difficult to record reliably" while " the direction of cardiac rate change is easily measured and appears to be at least as effective in distinguishing between the two systems (p. 137).

Based on her work using the cardiac response Graham proposed a third system of autonomic response, the startle reflex (SR), which would be elicited by transient stimuli with fast rise time and consists in

short latency cardiac acceleration that habituates quickly with repeated presentation of the stimulus.

The distinction between SR and DR was not confirmed by other authors. Turpin and Siddle (1978) found that high-intensity stimuli with slow rise time elicited a short-latency HR increment. According to Graham this cardiac response could be elicited only by fast rise time stimuli. Turpin and Siddle (1978) have considered "the possibility that startle and defense response are essentially the same" (p. 278).

Most Western studies have usually employed response windows of not more than 10 sec and have identified the short-latency HR acceleration occurring within this time after high-intensity auditory stimuli as DR. Turpin and Siddle (1978, 1981) have described a long-latency large cardiac response (HR acceleration of the order of around 25 beats with a peak latency of about 30 seconds) accompanied by an increase in forearm girth (an index of blood flow in the skeletal muscle) and digital and cephalic reduced vascular activity. Turpin (Turpin, 1986; Turpin and Siddle, 1978) has argued that this long-latency response is the one that Sokolov described as DR, and apparently Sokolov himself has agreed with this interpretation -Turpin (1986) refers to a Sokolov's personal communication. At variance with Sokolov's description of the DR, Turpin's long-latency cardiovascular response quickly habituates (it appeared only after the first stimulus of the series).

The two-component (short and long-latency) accelerative cardiac response to high-intensity stimuli has been found by other authors (e.g. Sartory, 1986, Fernandez, 1986). Bond (1943, cit. in Turpin and Siddle,

1978) found a similar pattern of cardiac response to sudden and intense stimuli in cats and dogs.

In summary, while low or moderate intensity stimuli elicit cardiac deceleration, which is concomitant with a receptive attitude of the organism toward the stimulation, high-intensity or aversive stimuli provoke a defensive reaction, which is accompanied by cardiac acceleration. Two components of this accelerative response have been described: a short-latency acceleration occurring shortly (onset latency of less than 2 seconds) after the stimulus, and a long-latency response taking place much later (peak latency of about 30 sec). The exact shape of the accelerative response may depend on the stimulus parameters (e.g. rise time, intensity), but the functional realtionship between stimulus charateristics and response shape is not well established yet. The assumption is that these cardiovascular responses "reflect the operation of a series of basic reflex mechanisms responsible for the initiation and control of sensory input" (Turpin, 1986, p. 1).

An important question is that of the nature, whether perceptuo-attentional or emotional, of these responses. For Sokolov and for Lacey as well as for Graham, the OR and DR systems have cognitive-attentional significance. However, the physiological response of the DR can also be interpreted within the context of Cannon's notion of the 'flight or fight reaction' (Cannon, 1929). The cardiovascular changes observed in the DR (i.e. cardiac acceleration and blood pressure increase) would be the necessary cardiovascular regulations demanded by the metabolic requirements in order to allow the organism to cope with threatening situations. Turpin (Turpin and Siddle, 1978, Turpin, 1986) has identified the accelerative cardiac response to high-intensity

stimuli with those cardiovascular reactions found in animals and humans when faced with aversive or stressing stimulation, and has viewed it as a component of the 'fight or flight' response, favouring, therefore, the emotional interpretation. However, the two interpretations, cognitive and emotional, are not necessarily incompatible.

The startle response has been found to be associated with chronic anxiety and fear reactions, which supports the emotional view. Hart (1974) found that anxious subjects showed greater increases in HR following a high-intensity tone than non-anxious subjects.

A paradigm which has been extensively used in animal research and has proved useful in the investigation of the anxiety reducing effects of numerous drugs is the so-called 'fear-potentiated startle paradigm', in which conditioned fear is measured "by an increase in the amplitude of a simple reflex (the acoustic startle reflex) in the presence of a cue previously paired with shock" (Davis, 1989, p. 71). This paradigm was developed on the basis of "anecdotal evidence that people startle more when they are afraid" (Davis, 1989, p. 53). In these studies muscle activity has been the usual measure of the startle reation.

The aim of the present experiment was to test the effects of alcohol on the psychophysiological response to high-intensity auditory stimuli. The SR/DR paradigm provides us with an experimental technique to study the reaction of the organism to intense/aversive stimuli. It has proven useful in the study of the anxiolytic effects of a number of drugs in animals (Davis, 1989). The study of the effects of alcohol on DR gives us the opportunity to examine the effects of alcohol on an early stage of the 'emotional cycle'. Subjects in this experiment will be presented

with a series of high-intensity auditory stimuli and the psychophysiological (HR and EDA) responses to these stimuli will be recorded. Although the cardiovascular response has normally been used in the study of the SR and DR, it has been suggested that aversive stimuli will produce longer recovery times of the skin conductance responses (Edelberg, 1973, Venables, 1987).

7.2 Methods

7.2.1. Subjects

The subjects were the same 22 male volunteers who took part in the experiment reported in chapter 6.

7.2.2. Design

There were two experimental groups, placebo and alcohol, with 11 subjects in each group. After the drinking and the absorption periods the subjects underwent a procedure to examine the psychophysiological startle and defense responses, in which ECG and EDA (SC) were recorded while they were exposed to tones of 105 db of intensity and two seconds of duration.

7.2.3. Apparatus

Five 1000 Hz tones were delivered through headphones at 65 seconds intervals. The tones were produced by a pattern generator and amplified to an intensity of 105 db. The tones, which had instantaneous rise and a duration of 2 seconds, appeared on a background of 40 db

white noise. The delivery of the tones was controlled by a BBC-B microcomputer. The pattern generator emitted a constant 1000 Hz tone and a filter controlled through the 'User-Port' of the BBC computer blocked the pass of the tone to the headphones except for the five 2-sec periods.

ECG and EDA were recorded by means of the same apparatus and following the same procedure as in chapter 6. Psychophysiological recording started 10 seconds before the onset of the tone and continued for 50 seconds after the end of each stimulus. A relatively long baseline period of 10 seconds was used in order to control for sinus arrhythmia.

7.2.4. Dose and placebo manipulation

Subjects in the alcohol group received 0.8 g of ethanol per kg of body weight. All subjects were told that the objective of the experiment was to compare the effects of two doses of alcohol.

7.2.5. Procedure

After consuming the beverage (see chapter 6 for details of administration) an absorption period of 25 min was allowed. Then the subjects completed the mood inventories and scales (as described in chapter 6) and approximately 30 minutes after the consumption of the drink the procedure to test the startle and defence responses started. Subjects were told that the purpose of the procedure was to test the physiological response evoked by tones of different frequency and intensity and informed that they would hear several tones at different intervals through the headphones. After the subject had put the

headphones on, three minutes were allowed before the first tone was generated.

7.2.6. Response quantification

The processing of skin conductance data was similar to the one carried out in chapter 6. Response amplitude, latency, rise time and 50% recovery time were scored by hand from the skin conductance trace plotted with a resolution of 4.2 cm per micromho and 3.5 mm per second. These measures were taken from the evoked skin conductance responses (SCR), which were defined as an increase in skin conductance greater than 0.05 micromhos taking place between 1 and 5 sec after stimulus offset. Amplitude was measured to the nearest millimetre from the level prior to the stimulus onset to peak increase. The resolution of the amplitude measures was +/- 0.023 micromhos. Temporal variables were measured to the nearest millimetre, which gave a resolution of +/- 0.28 seconds. Latency was measured from stimulus onset to response onset. Rise time was calculated as the time from response onset to the point of maximum amplitude. '50% recovery time' was measured as the time from peak response to the point in which the skin conductance had returned to 50 per cent of the maximum increase.

ECG data were analyzed off-line by means of a computer program that detected QRS peaks, measured cardiac periods and calculated HR second by second, from 10 seconds before stimulus onset to 50 seconds after stimulus offset. The average HR of the 10 pre-stimulus seconds was calculated and differential scores were computed by subtracting the mean pre-stimulus HR from the HR values of each second after stimulus onset.

Short and long latency evoked responses were scored following criteria adapted from Turpin and Siddle (1978). A short-latency evoked cardiac response was defined as an increase of HR of at least 2 beats per minute greater than the highest HR observed during the 9 seconds (although 10 seconds were recorded it is impossible to calculate the HR of the first second) prior to stimulus onset, occurring within the first five seconds after the onset of the stimulus. The criteria for scoring the long-latency evoked cardiac responses were that there was an increase in HR of at least 10 beats per minute about the average pre-stimulus HR maintained for at least 3 seconds.

7.3. Results

The data of the amplitude of the SCRs (given in table 7.1.) were analyzed by means of 2 x 5 (group x tone) ANOVA, with the first factor varying between subjects and the second within subjects. The program BMDP P4V (Dixon, 1985) was used for this and all the other ANOVAs conducted in this chapter. There was no difference between groups. The reduction of amplitude over presentations followed a linear trend in both groups.

Only two subjects (one in each group) failed to emit a SCR to the first tone, but the number of non responders increased gradually and the SCR was absent in seven subjects (three in the alcohol group and four in the placebo group) after the fifth tone. Time variables were analyzed by means of non-parametric tests. Mann-Whitney tests were used to compare the two groups at each stimulus. Data of latency, rise time

and 50% recovery time are given in tables 7.2., 7.3. and 7.4 respectively. No difference between groups was found on latency or rise time. Recovery time in the SCR evoked by the first stimulus was significantly larger in the placebo group (Mann-Whitney U=23, p=0.04). This difference disappeared after the first tone (see table 7.4.).

In order to analyze the short-latency cardiac response, the second-by-second HR differences from pre-stimulus level during the 10-sec period after stimulus onset were examined. The placebo group showed a clear decelerative response while the alcohol group did not (see fig 7.1a.). Fig 7.1b. shows the number of evoked cardiac responses to each tone during this period. The number of short-latency cardiac responses evoked by the first tone was significantly larger in the alcohol group (chi-square= 4.05, df=1, $p<0.05$). HR changes from baseline during this period after the first tone were analyzed by means of 2(group) x 10 (time) ANOVA. The effect of group was nonsignificant (F=1.96, df=1/19, p=1.17). Neither time nor the interaction influenced the cardiac response either (respectively, F=1.57, df=3.07/58.25, p=0.20 and $F<1$).

Fig 7.2. shows the second-by-second cardiac response (difference from the average HR in the pre-stimulus period) during a period of 50 seconds after stimulus onset. No long-latency accelerative response can be observed in either group. No effect of group, time, or the interaction was found in a 2 (group) x 40 (time) ANOVA performed on the HR changes from minute 11 to minute 50 of the post-stimulus period. Only the responses to the first tone in four subjects (two in each group) met the criteria for long-latency evoked cardiac response.

Table 7.1. SCR amplitude -log (micromhos)+100- of evoked responses to 105 db tones. Means (and standard deviations).

	alcohol (n=11)	placebo (n=11)
Tone 1	1.73 (0.63)	1.79 (0.64)
Tone 2	1.49 (0.60)	1.48 (0.59)
Tone 3	1.29 (0.70)	1.41 (0.61)
Tone 4	1.24 (0.71)	1.14 (0.64)
Tone 5	0.92 (0.77)	0.99 (0.68)

Table 7.2. Latencies (in seconds) of SCRs to 105 db tones. Means (and standard deviations).

	alcohol	n	placebo	n	p
Tone 1	2.03 (.43)	10	2.17 (.40)	10	n.s.
Tone 2	1.77 (.56)	9	2.11 (.72)	10	n.s.
Tone 3	1.81 (.65)	9	1.71 (.66)	8	n.s.
Tone 4	1.85 (.73)	8	1.92 (.65)	7	n.s.
Tone 5	2.11 (.50)	8	2.20 (.21)	7	n.s.

Table 7.3. Rise time (in seconds) of SCRs to 105 db tones. Means (and standard deviations).

	alcohol	n	placebo	n	p
Tone 1	3.91 (1.97)	10	4.48 (1.95)	10	n.s.
Tone 2	2.70 (0.73)	9	2.91 (1.12)	10	n.s.
Tone 3	2.47 (0.78)	9	3.10 (1.46)	8	n.s.
Tone 4	2.50 (0.54)	8	2.41 (1.06)	7	n.s.
Tone 5	1.82 (0.37)	8	2.16 (0.63)	7	n.s.

Table 7.4. 50% recovery time (in seconds) of SCRs to 105 db tones. Means (and standard deviations).

	alcohol	n	placebo	n	p
Tone 1	13.02 (8.28)	10	30.97 (18.64)	10	0.04
Tone 2	5.20 (2.61)	9	6.71 (5.85)	10	n.s.
Tone 3	4.44 (3.04)	9	7.85 (5.70)	8	0.09
Tone 4	7.28 (4.88)	8	8.57 (6.05)	7	n.s.
Tone 5	5.71 (2.99)	8	4.61 (1.92)	7	n.s.

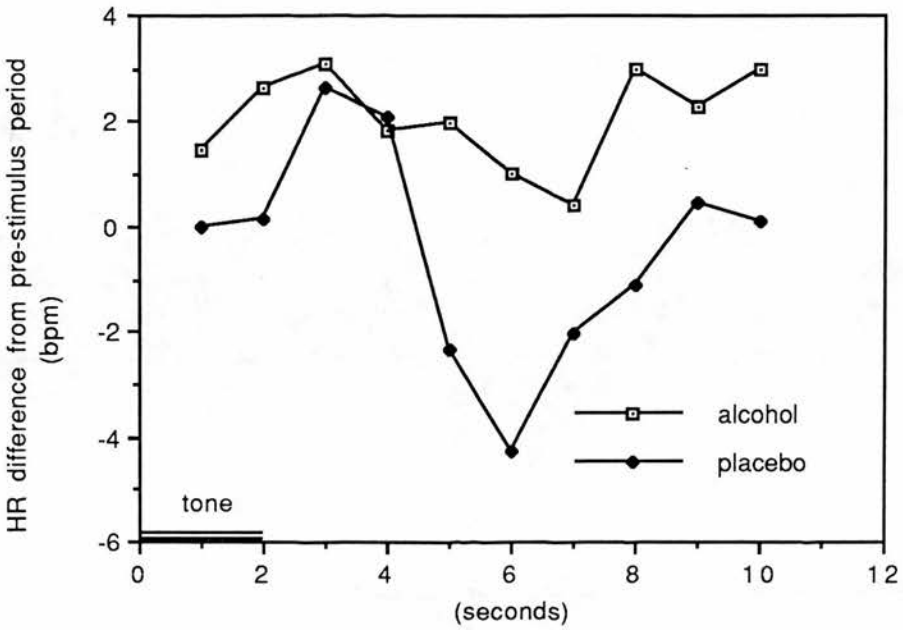

Fig 7.1a. Mean HR changes from pre-stimulus level in the 10-sec period after stimulus onset.

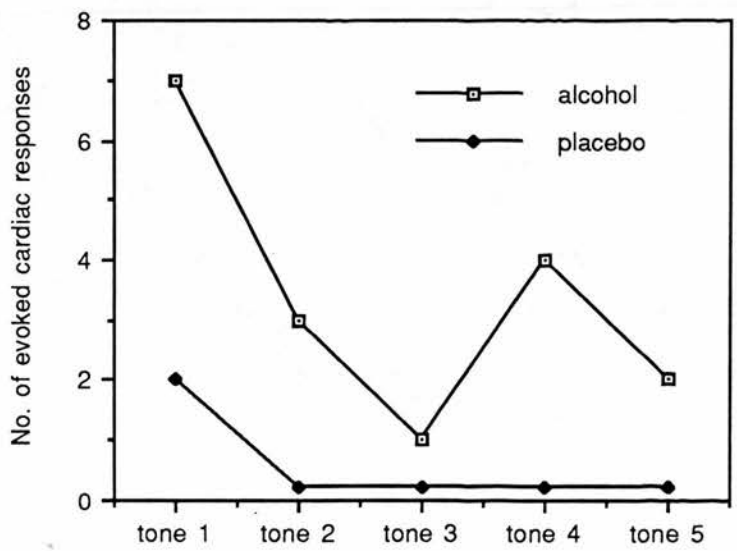

Fig 7.1b. Number of short-latency evoked cardiac responses to 105 db tones in alcohol and placebo groups.

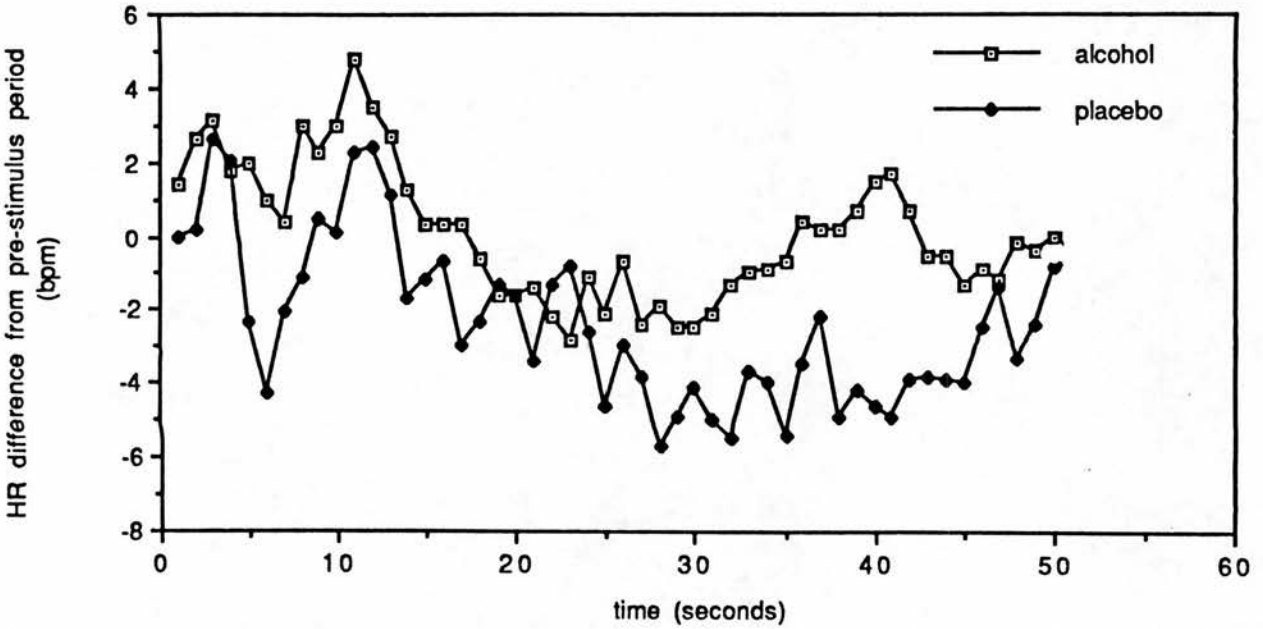

Fig 7.2. HR changes from pre-stimulus level, second-by-second, for a 50-sec period after stimulus onset.

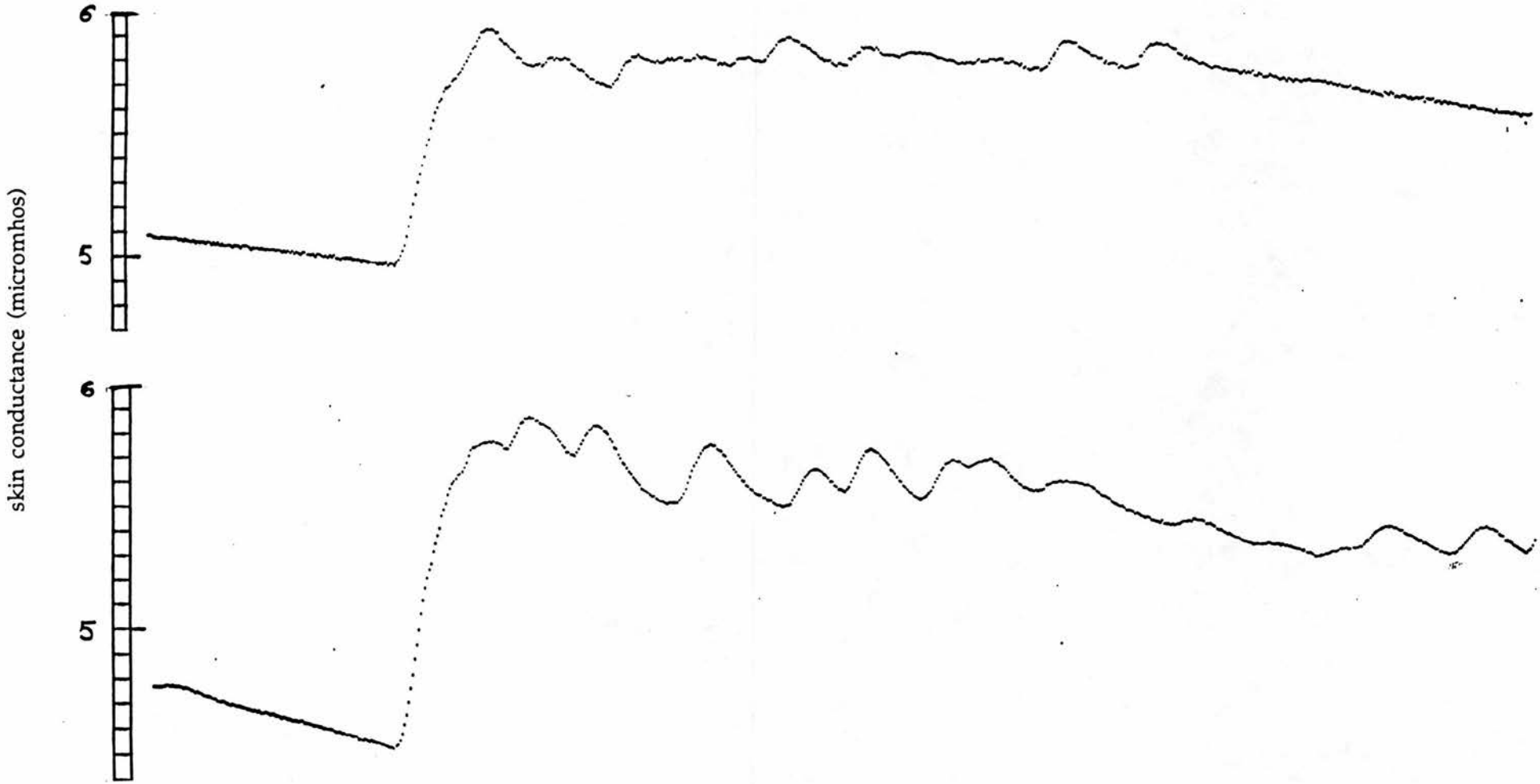

Fig 7.3. Examples of skin conductance response to the first tone in two subjects in the placebo group.

7.4. Discussion

It has been suggested (Venables, 1987) that while the amplitude of the SCR does not differentiate between OR and DR the recovery time is longer in the latter. Long recovery times in response to intense auditory stimuli have been found by Dimberg (1990). The results of the present experiment are apparently in accordance with this. Recovery time of the SCR to the first stimulus was longer in the placebo group. This larger response quickly habituated so there was no difference between groups for the following four stimuli. This is in agreement with the results reported by Turpin and Siddle (1978) who found a defence response only after the first stimulus.

The stimuli used in this experiment did not elicit the long-latency cardiac response, which has been proposed by Turpin (1986) as the real Sokolov's DR. Sokolov has suggested that painful stimuli are necessary to elicit the defense response. Turpin and Siddle (1978), on the other hand, found the long-latency response using white noise of 110 db, and white noise seems to be more stressing than pure tones (Turpin, 1986). Therefore it seems possible that the 1000 Hz, 105 db tones used in this experiment were not sufficient to produce the long-latency component of the cardiac defence response.

The short-latency cardiac responses seem to indicate that alcohol subjects were more startled, whereas the subjects who did not drink alcohol seem to have reacted in a more receptive, 'orienting response' way to the stimulus.

Turpin (1986, Turpin and Siddle, 1978) proposed that the short-latency (in Graham's terms the SR) and the long-latency responses are the same response. However, the long-latency component seems to require stronger, more stressing stimulation. One may hypothesize a continuum in the receptive-defensive attitude or response of an organism towards stimulation. A non defensive, receptive attitude would produce an orienting response. A moderately anxious or defensive attitude would generate a startle response with short-latency cardiac acceleration. A defense response (in the strict Sokolov's conception) or 'flight or fight' response, with long-latency cardiac acceleration, would appear only in extremely anxious states or in response to very noxious or aversive stimuli. Anxiolytic drugs would reduce both defensive responses. In the present study, the difference between groups in the short-latency cardiac response to the first tone seems to indicate that the alcohol group reacted in a more defensive manner.

The longer recovery times of the SCRs to the first tone seem to challenge this interpretation. However, further inspection of skin conductance traces suggests the possibility that the longer recovery times were caused by the occurrence of non-specific responses following the first skin conductance response. Fig. 7.3 shows examples of this phenomenon. In a recent study designed to examine the psychological correlates of non specific electrodermal activity, Nikula (1991) concluded that cognitive processes (e.g. inner speech) seem to underlie the non specific skin conductance responses. Thus, the longer recovery times might be interpreted as the indication of a higher degree of cognitive processing triggered by the stimulus. This would be in accordance with the results of the cardiac response. In the light of the continuum in the

receptive-defensive attitude towards stimulation I have hypothesized it could be interpreted that alcohol made the subjects react to stimulation in a less receptive way. It is possible, however, that this effect might have been caused by the blood alcohol concentration still increasing at the moment of the test. Further research should investigate the response at different points of the BAC curve.

Chapter 8.
THE ACTION OF ALCOHOL ON THE RECOGNITION OF EMOTIONAL EXPRESSION

8.1. Introduction

Several authors (e.g. Hull, 1981, Steele and Southwick, 1985, Tucker and Vuchinich, 1983, Yankofsky et al, 1986) have hypothesized that the possible effects of alcohol on anxiety are secondary to the action of alcohol on the cognitive processes which determine the affective response. According to this theory, "rather than modifying affect (e.g. anxiety) directly, alcohol is assumed to influence a complex set of cognitive-perceptual processes that then lead to emotional reactions. Tension reduction, in this framework, is an indirect result of primary changes in cognitive-perceptual processing of information" (Yankofsky et al 1986, p. 26).

Keaman and Lisman (1980), in a study already reviewed in chapter 4, found some evidence suggesting that alcohol may interfere with the evaluation of one's own behaviour and its impact on others during a social interaction. This effect was, however, found only in a sample of shy men, but not in normals. Yankofsky et al (1986) designed a study to examine the effects of alcohol on perception of interpersonal feedback and self-evaluation, assuming that the anxiety response depends upon these processes. They used a BPD in an experiment in which male undergraduates interacted with a female confederate at two points: before drinking (either a placebo or a beverage containing a dose of 0.75 g/kg) and after consumption. The first time, the confederate was friendly and responsive; the second time, the

interaction took place under "negative feedback" conditions, e.g. the confederate "responded to questions only minimally", "broke no silences", etc. After each interaction the subjects viewed the videotapes of their performance and rated them. The subjects were also asked to rate their self-perceptions before the interactions and after the rating of the videotapes. Yankofsky et al. (1986) found that self-perceptions of control and power as measured by semantic differential ratings were unaffected by the negative feedback in the intoxicated subjects, whereas they were diminished in those who had the placebo drink. In addition, those who did not drink alcohol rated their performance, when they viewed the videotapes of the interaction, more negatively than those who drank alcohol. The belief of having drunk alcohol did not affect the main dependent variables. The videotapes of the interactions were also rated by a group of judges. No differences were found between the first and the second interactions, or amongst the four experimental groups. When viewing the videotapes, the subjects were instructed to stop the tape every time they judged their behaviour to be positive or negative. Alcohol did not influence the number of evaluations the subjects made. Self-reported anxiety was also unaffected by the experimental factors. Yankofsky et al (1986) interpreted the results of this experiment as indicating that alcohol interferes with the perception of negative feedback. In the paradigm used by Yankofsky et al (1986) it is not clear which processes alcohol influenced. It is not possible to conclude whether alcohol influenced the perception of interpersonal cues or the interpretation of these signals, or whether under the effects of ethanol the subjects did not pay attention to the relevant stimuli.

Tucker and Vuchinich (1983) have advocated the direct study of the

effects of alcohol on more primary processes relevant for the effective processing of interpersonal social information. They used a BPD in a study in which the effects of alcohol on the perception of facial expressions of emotion were investigated. They found that those who received alcohol (0.55 g/kg the males and 0.50 the females) made more recognition errors than those who did not, although this difference was only statistically significant in a one-tailed comparison. The greatest impairment occurred when subjects both consumed alcohol and were told that their drinks contained alcohol.

In a study in which the effects of alcohol, marijuana or placebo on recognition of emotions conveyed by the face were investigated, O'Sullivan (1975, cit in Borrill et al, 1987) found that subjects who drank alcohol (0.8 g/kg) made significantly more mistakes in the recognition of anger and sadness.

Apparently without knowing Tucker and Vuchinich's (1983) paper, Borrill et al (1987) designed an experiment to examine the action of alcohol on perception of facial expressions of emotion. Subjects (males and females) drank either a placebo or one of two doses (0.79 g or 1.975 g of vodka/kg) prior to the experimental task in which they were asked to identify pictures of faces expressing six basic emotions (happiness, surprise, fear, anger, sadness, disgust). The low dose slightly improved performance while those who consumed the high dose made more mistakes in the categorization task. The main effect of alcohol on total number of correct responses was statistically significant, as was the pairwise comparison (by Scheffe's method) between low and high doses. The contrast between placebo and high dose was not statistically significant. When the recognition of each emotion was

analysed separately, the main effect of alcohol was statistically significant for anger and disgust. Pairwise comparisons were not reported. However, an examination of their figures suggests that statistical significance was due to the combination of both improvement caused by the low dose and impairment produced by the consumption of the high dose.

The purpose of the present experiment was to investigate further the possibility that alcohol impairs the recognition of emotional expressions. It was also of interest to discover whether this effect is confined to facial expressions, and therefore a test of recognition of emotion as conveyed by the voice was included in the study in addition to a task of categorization of facial expressions of emotion.

The comparison between the effects of alcohol on recognition of facial and oral expressions of emotion will also allow us to decide whether the nature of these effects is emotional or cognitive-perceptual. If it is found that the action of alcohol is specific to visually expressed emotional information, this would lead to the conclusion that the effects of alcohol on the perception of emotion conveyed by facial cues is secondary to a disruption of visual perception. Alternatively, alcohol may reduce accuracy in the recognition of emotion expressed through both media, visual and auditory, and this would suggest a more general cognitive impairing effect or an impairment in emotional processing.

The difficulty in recognizing a particular emotion depends on the medium of presentation. In a study which used the same oral material as the present experiment and also pictures from Ekman and Friesen

(1976), happiness was found to be the most easily recognizable emotion when expressed in the face, but the most difficult one when communicated vocally (Law, 1985). Therefore, if alcohol intoxication is shown to impair the same emotions in both media, one may deduce that alcohol affects the emotional processing of specific emotions, and, hence, that its effect is of an emotional nature.

The nature of the effects of alcohol on the perception of expression of emotion was further investigated by administering a test designed to measure visuo-spatial perceptual abilities involved in the processing of faces, the Benton Test of Facial Recognition (Benton et al, 1978). A positive correlation between the scores on this test and the accuracy measures in the recognition of facial cues of emotion, accompanied by no relationship between recognition of orally expressed emotion and the scores on the Benton test, would be expected if alcohol primarily affects visuo-spatial perception.

8.2. Method

8.2.1. Design

The effects of a moderately high dose of alcohol on measures of perception of expressive interpersonal stimuli were investigated using a between subjects design. Half of the subjects drank a placebo beverage and the other half consumed an alcoholic drink. Subsequently, the subjects were tested on the recognition of oral and facial expression of emotion and on the perception of facial identity.

8.2.2. Subjects

Thirty males were allocated randomly to either the placebo or alcohol conditions. Volunteers were recruited through notices placed around the University campus. Only male social drinkers who did not suffer from any health condition incompatible with alcohol consumption were accepted in the study. Teetotallers and individuals with drinking or psychiatric problems were also excluded. The subjects taking part in the experiment were mostly undergraduate students with a mean age of 21.2 yrs (SD=5.56). They reported a weekly average alcohol consumption of 26.7 units (SD=16.22) distributed throughout a mean of 2.26 drinking occasions per week (SD=0.58). At the first contact subjects were given information about what the experiment involved. If they agreed to take part in the study they were asked to comply with the following requirements prior to the experimental session: (1) to fast for three hours; (2) not to drink any alcohol for 12 hours; and (3) not to take any drug or medicine for 12 hours. The subjects were paid three pounds for their participation.

8.2.3. Materials

Recognition of facial and oral expression of emotion

Thirty six photographs (six for each basic emotion: happiness, fear, sadness, disgust, anger and surprise) from the set prepared by Ekman and Friesen (1976) were used as stimuli. Six pictures for each basic emotion, three displayed by a male and three by a female, were chosen from the 110 photographs compiled by Ekman and Friesen (1976).

Those with the highest interrater reliability were selected.

The oral stimuli consisted of the set of recorded sentences developed by Law (1985). Law (1985) asked two amateur actors, one male and one female to recite two sentences without any intrinsic emotional content ("I will be away for a long time" and "When will they come back again?") in a way which expressed each of the six basic emotions through tone of voice. Law (1985) conducted a pilot study where 15 subjects, nine females and six males, were asked to identify the emotion expressed in each sentence. According to the information Law (1985) provides, each sentence in the set of 24 was recognized as expression of the intended emotion by at least 68% of the judges, and seven were recognized by 100%.

The photographs and sentences were recorded on a videotape in a random order. Each item, either picture or sentence, was preceded by an auditory tone and the appearence of the corresponding item number on the TV screen. This number remained on the screen for a second until the appearance of the experimental stimulus. The exposure time of the photograph showing the facial expression was one second. Between the end of one of the stimuli and the cue for the next there was an interval of six seconds. Subjects were asked to match each of the stimuli to one of the six basic emotions (happiness, sadness, fear, disgust, anger and surprise).

A "Panasonic NV-180 Portable Cassette Recorder" and a "Panasonic" 11 inches colour TV monitor were used for the administration of this test.

Recognition of facial identity

The short form of the 'Test of Facial Recognition' (Benton et al, 1978) was used to assess perception of faces without emotional component. In this test, the subject is presented with a front view of a face and asked to say which of six photographs on the stimulus card represents the same person as the sample. The test includes three kinds of stimulus cards: front views, front views and side views or front views under different lighting conditions. This test is said to be a "standardized objective procedure for assessing the capacity to identify and discriminate photographs of unfamiliar faces". Clinical evidence suggests that this task has a "substantial visuo-spatial processing component" (Lezak, 1983, p.352).

Mood was monitored throughout the experimental session using the "Mood Adjective Checklist" (Mackay et al, 1978).

Visual Analogue Scales were used to measure perceived intoxication. They consisted of 100-mm horizontal lines anchored on each side by the terms "Totally sober" and "Totally drunk".

8.2.4. Dose and placebo manipulation

Subjects in both groups, placebo and alcohol, were told that their drinks contained alcohol. Those subjects allocated to the alcohol group consumed a dose of 0.8 gr of pure alcohol per kg of body weight. Ninety per cent pure ethanol was used. It was diluted in a mix of bitter lemon and Rose's lime juice at a proportion of 1:9:1 (alcohol:bitter

lemon:lime juice). The placebo group received only the "soft" part of the mix.

8.2.5. Procedure

When the subject arrived at the laboratory he received information about what the experiment involved and was required to sign a consent form (see Appendix). All subjects were told that the aim of the experiment was to investigate the effects of different dose levels of alcohol, and that, therefore, different individuals would receive different amounts of alcohol. The subject was weighed and asked to complete the mood inventory (MACL). The experimenter brought a jug containing the drink and divided it into six equal parts. The subject was instructed to consume each glass within three minutes, which resulted in a total consumption time of 18 minutes. During the consumption (18 minutes) and absorption (25 minutes) periods, the subject was left on his own and provided with newspapers and magazines. The experimenter checked that the drinking instructions were followed adequately several times during the consumption period. Twenty five minutes after finishing the drink, the subject completed the MACL again. He was also asked to indicate on a VAS how drunk he felt. Then, the "Test of Recognition of Facial and Oral Expression of Emotion" and the Benton "Test of Facial Recognition" were administered consecutively.

For the "Test of recognition of facial and oral expression of emotion", the subject was instructed as follows:

" This test consists of items of two sorts: photographs of

people and spoken sentences. In both cases the persons were asked to express different emotions. Your task will be to judge WHAT EMOTION is shown by the person, either through the voice or by the facial expression. One of these six emotions may have been expressed: happiness, sadness, fear, disgust, anger and surprise.

Try to regard each photograph or sentence as a totally new task and try not to be influenced by your previous judgments.

The content of the sentences, that is, the actual words included in them, will be the same for most of them. You will have to pay attention to how they are told. The different emotions will be expressed in the tone.

You will see each picture or hear each sentence only once, and will have only a few seconds to answer. So you must work quickly. Speak out your answer clearly so that the experimenter takes note of it. If you are uncertain about a picture or sentence, make a guess. Follow your first impression.

Give only one answer for each picture or sentence. You may think that more than one emotion is expressed in the picture or sentence, but you must choose the one word from the six emotional categories listed that you feel best describes the emotion the person is showing."

The subject sat in an easy chair at a distance of 1.75 m from the TV set. A sheet with the list of the six basic emotions was visible all the time under the TV set.

Benton's Test of Facial Recognition was administered as specified in the manual. The test stimuli are contained in a spiral bound booklet. The sample photograph and the answer alternatives are presented on facing pages, the subject seeing the target picture on top of the response choices. The Short Form of this test consists of 13 items. For the first six, the experimenter points to the sample photograph and asks the subject to identify which one of the six response pictures shows the same person. In the remaining seven items, the person in the sample photograph is shown in three of the choice pictures, and the subject is instructed to indicate which three they are.

After the completion of the experimental tests, the subject was asked to complete the mood inventory for the third time and to indicate how drunk he felt using a VAS. The subject was also asked to estimate how much alcohol he had drunk.

Finally, the subject was debriefed about the content of the drink and the design and the object of the experiment, and was given the three pounds.

8.3. Results

8.3.1. Checks of the placebo manipulation

It could be said that placebo manipulation was succesful in that no

subject in the placebo group indicated any suspicion or disbelief that they had consumed alcohol. Moreover, when, at the end of the experiment, they were debriefed about the real content of their drinks, the most common reaction was surprise. A more solid index of success of the placebo manipulation was that all placebo subjects, when asked to guess how much alcohol they had drunk, estimated a consumption ranging from the equivalent to a pint to three pints of beer (Mean=1.9, SD=0.573), while on the same question subjects in the alcohol group gave significantly higher estimates of consumption (Mean=3.467, SD=1.187, t=4.602, df=20, $p<0.0001$). The analysis of perceived intoxication ratings also reflects a greater degree of reported intoxication by the alcohol group. A 2x2 ANOVA was performed on data of perceived intoxication (these data are given in table 8.1.) in order to investigate the effects of group (alcohol and placebo) and time (30 and 60 min after consumption). There was a statistically significant effect of treatment group ($F(1,28)=5.73$, $p=0.023$). The effect of time and the group-time interaction were non-significant.

8.3.2. Mood

Arousal and stress scores from the MACL were transformed into percentage scores. This transformation yielded scores ranging from 0 to 100 for both arousal and stress. Two 2x2 ANOVAs (program BMDP P4V, Dixon, 1985) were used to analyze the effects of alcohol on subjective stress and arousal after drinking, where group (placebo vs alcohol) was the between subjects variable and time (30 and 60 minutes after finishing the consumption) was the within subjects factor. Dependent variables were change scores, i.e. observed post-drinking scores minus the baseline scores recorded at the start of the

experimental session. In order to eliminate negative values an arbitrary figure (100) was added to the differentials before the analysis so that a transformed change score of 100 stands for no change from baseline levels. Means and standard deviations of these data are presented in table 8.2.. Stress was reduced to a similar degree in both groups. This effect can be observed 30 min after the consumption and remains about the same level half an hour later. Alcohol exerted no effect on subjective stress ($F<1$), nor did time ($F<1$). Thirty minutes after consumption, arousal was reduced in the placebo group whereas a slight increase can be observed in those who consumed alcohol. At the end of the experimental session subjective reports of arousal had dropped markedly in the alcohol group and approached placebo group levels, which had diminished slightly. The 2x2 ANOVA yielded an almost significant main effect of group (alcohol vs placebo) ($F(1,28) = 3.90$, $p = 0.058$). Neither the main effect of time nor the interaction effect were statistically significant (in both cases $F<1$). In the simple effect analyses the effect of time for the alcohol group was close to statistical significance ($F(1,28) = 4.05$, $p= 0.054$). No other effect approached statistical significance.

8.3.3. Recognition of facial and oral expression of emotion

Two dependent variables were extracted from these tests: (1) the number of times each emotion label was attached to any stimuli, which we shall call 'emotion responses', and (2) the number of 'correct recognitions' of each emotion. 'Emotion responses' to oral and facial expressions were computed separately and transformed into percentage scores in order to make comparisons between them possible. Response scores for the facial part of the test were divided

by 6 (the number of facial stimuli per emotion) and multiplied by 100; in the case of responses to oral expressions the divisor was 4 (as there were 4 sentences expressing each emotion). For correct recognition scores, percentages were also calculated, and subsequently a correction for response bias was computed according to the formula:

$$\%CR' = (\%CR + (\%CR / \%R)) / 2$$

where %CR' = percentage of correct recognitions corrected for response bias; %CR = percentage of correct recognitions; and %R = percentage of 'emotion responses' (Van Bezzoyen, 1984).

Emotion responses

The data of emotional responses are summarized in table 8.3.. A 2x2x6 ANOVA (program BMDP P4V, Dixon, 1985) was performed on these data. This analysis included a between subjects factor, group, and two repeated measures factors, medium of expression (oral and facial) and emotion. Of course, no effect of medium or group will be found as the means for both groups and for both media are necessarily identical. A statistically significant effect of emotion was found (Greenhouse-Geisser Adj df $F(3.34, 93.58) = 6.87$, $p = 0.0002$) indicating that there was a bias in the responses of the subjects to the different emotions. The interaction between group and emotion was also statistically significant (Greenhouse-Geisser Adj df $F(3.34, 93.658) = 2.77$, $p = 0.04$) reflecting that each group showed different patterns of emotion-biased responses. When analysis (2x6 ANOVA) was performed for medium separately, results revealed that, although the

emotion influence on responses was statistically significant for both oral and facial recognitions (respectively, Greenhouse-Geisser Adj df F(3.23, 90.5) = 8.04, p = 0.0001 and Greenhouse-Geisser Adj df F(3.24, 90.6) = 7.41, p = 0.0001), the interaction between group and emotion was significant only for responses to facial expressions. (Interaction at 'oral' level: Greenhouse-Geisser Adj df F(3.23, 90.5) = 0.52, p = 0.68; at 'facial': Greenhouse-Geisser Adj df F(3.24, 90.6) = 4.66, p = 0.0036). Therefore, alcohol seems to affect the emotion-biased pattern of responses only on the recognition of facial expression.

In order to examine further how group (between subjects factor) and medium (repeated measures variable) influenced the frequency in which subjects responded with each emotion label, a series of 2x2 mixed ANOVA were performed. Results of these ANOVAs are presented in Table 8.4.. There was a statistically significant effect of medium for five of the six emotions. Only the effect on 'fear' responses did not reach statistical significance, F(1,28) = 3.06, p = 0.091. Group, that is, whether subjects had drunk alcohol or not, significantly influenced the endorsement of the label 'surprise' to the presented emotional expressions, F(1,28) = 11.7, p = 0.001. Those who received an alcoholic drink used the 'surprise' label more frequently than those who were administered the placebo. The analysis of simple effects revealed that the group factor influenced 'surprise' responses to facial expressions (F(1,28) = 9.94, p = 0.003) but did not influence subjects responses to oral expressions (F(1,28) = 1.34, p = 0.257). Although the main effect of group on 'fear' responses was not statistically significant (F(1,28) = 2.65, p = 0.115), when responses to facial and oral expressions of emotion were analyzed separatedly, it became apparent that alcohol reduced the number of 'fear' responses

to facial expressions ($F(1,28) = 11.73$, $p = 0.0019$) and that 'fear' responses to oral expressions were unaffected ($F<1$).

Correct Recognition

Percentage of correct recognitions, once corrected for response bias (data summarized in table 8.5.), was analyzed by means of a 2x2x6 ANOVA with alcohol as a between subjects variable and medium and emotion as repeated measures factors. As a whole, facial expressions of emotion were recognized more easily than oral expressions, and this was reflected in a statistically significant main effect of medium, $F(1,28) = 18.02$, $p = 0.0002$. Emotion also produced a significant main effect (Greenhouse-Geisser Adj df $F(4.41, 123.5) = 11.93$, $p<0.0001$). Group did not yield a statistically significant effect ($F<1$), nor did the interaction group x medium ($F(1,28) = 2.49$, $p = 0.125$). The interaction between emotion and group approached conventional levels of statistical significance (Greenhouse-Geisser Adj df $F(4.41, 123.5) = 2.32$, $p = 0.054$). When the analysis was performed at each level of the 'medium' factor separately, the effect of emotion was still significant at both levels of the variable (at the 'oral' level, Greenhouse-Geisser Adj df $F(4.10, 111.78) = 7$, $p<0.0001$; at the 'facial' level, Greenhouse-Geisser Adj df $F(3.73, 104.45) = 14.64$, $p<0.0001$). There was no effect of treatment group at the 'oral' level. Subjects who did not drink alcohol tended to perform better on the recognition of facial expressions of emotion, although this effect was only a statistical trend ($F(1,28) = 3.10$, $p = 0.089$). While there was no emotion x group interaction at the 'oral' level (Greenhouse-Geisser Adj df $F<1$), this interaction effect was clear on the recognition of facial stimuli, (Greenhouse-Geisser Adj df $F(3.73, 104.45) = 3.86$, $p = 0.0069$).

Table 8.1. Perceived intoxication (100 mm VAS) 30 min and 30 min after consumption. Group means (and standard deviations).

	placebo (n=15)	alcohol (n=15)
After 30 min	21.46 (3.53)	32.06 (4.51)
After 60 min	15.93 (2.75)	29.46 (4.79)

Table 8.2. Change scores of arousal and stress (MACL) 30 and 60 min after consumption. Means (and standard deviations).

	placebo (n=15)		alcohol (n=15)	
	arousal	stress	arousal	stress
After 30 min	96.66 (6.68)	91.97 (3.48)	106.29 (5.46)	95.30 (2.81)
After 60 min	93.14 (5.68)	94.19 (3.53)	97.22 (4.15)	92.83 (2.66)

Table 8.3. 'Emotion responses' emitted to the presentation of each category of oral and facial stimuli in the alcohol and placebo groups. Raw frequencies have been transformed by dividing frequencies of responses to oral stimuli by four, and frequencies of responses to facial stimuli by six, and multiplying in both cases by 100, in order to make the oral (with four stimuli category) and the facial (with six stimuli for category) parts of the test comparable.

	placebo (n=15)		**alcohol (n=15)**	
	stimuli			
	oral	facial	oral	facial
happiness	81.66 (34.67)	106.66 (10.54)	75.00 (36.59)	102.22 (5.86)
sadness	133.33 (45.96)	97.77 (12.38)	130.00 (45.51)	91.11 (27.36)
fear	98.33 (52.15)	93.33 (23.40)	96.66 (50.76)	63.33 (24.55)
anger	131.66 (31.99)	98.89 (26.32)	116.66 (37.40)	103.33 (24.55)
surprise	78.33 (46.16)	100.00 (19.91)	98.33 (48.61)	132.22 (34.19)
disgust	71.66 (29.68)	103.31 (24.57)	78.33 (36.43)	107.77 (30.12)

Table 8.5. Correct recognitions corrected for respoonse bias of oral and facial expressions of emotion in the alcohol and placebo groups. Means (and standard deviations).

	placebo (n=15)		**alcohol (n=15)**	
	stimuli			
	oral	facial	oral	facial
happiness	36.28 (13.23)	50.47 (0.04)	32.95 (14.78)	50.49 (0.02)
sadness	45.70 (6.78)	47.14 (5.28)	45.36 (11.40)	41.56 (12.41)
fear	32.79 (17.04)	40.44 (7.84)	32.85 (13.24)	27.09 (11.91)
anger	42.83 (7.96)	39.85 (9.73)	42.03 (10.22)	40.39 (8.48)
surprise	30.42 (12.27)	42.65 (8.03)	37.92 (11.53)	44.79 (8.76)
disgust	30.28 (12.27)	42.65 (8.03)	37.92 (11.53)	44.79 (8.76)

Table 8.4. 2 x 2 ANOVAs on 'emotion responses' analysing each emotion separately.

EMOTION	source	d.f.	F	p
happiness	group	1,28	<1	
	medium	1,28	15.36	0.0005
	g x m	1,28	<1	
sadness	group	1,28	<1	
	medium	1,28	16.99	0.0003
	g x m	1,28	<1	
fear	group	1,28	2.65	0.115
	medium	1,28	3.06	0.091
	g x m	1,28	1.66	
anger	group	1,28	<1	
	medium	1,28	7.52	0.01
	g x m	1,28	1.34	
surprise	group	1,28	11.70	0.001
	medium	1,28	5.37	0.028
	g x m	1,28	<1	
disgust	group	1,28	<1	
	medium	1,28	11.06	0.0025
	g x m	1,28	<1	

p values are reported only if < 0.1 or approaching this level.

Table 8.6. 2 x 2 ANOVAs on percentage of correct recognitions (after corrected for response bias), analysing each emotion separately.

EMOTION	source	d.f.	F	p
happiness	group	1,28	<1	
	medium	1,28	38.38	0.0001
	g x m	1,28	<1	
sadness	group	1,28	1.82	0.187
	medium	1,28	<1	
	g x m	1,28	<1	
fear	group	1,28	3.69	0.064
	medium	1,28	<1	
	g x m	1,28	4.33	0.046
anger	group	1,28	<1	
	medium	1,28	1.28	
	g x m	1,28	<1	
surprise	group	1,28	3.18	0.085
	medium	1,28	13.26	0.0011
	g x m	1,28	1.04	
disgust	group	1,28	<1	
	medium	1,28	7.43	0.01
	g x m	1,28	<1	

p values are reported only if < 0.1 or approaching this level.

Table 8.7. Frequency of 'emotion response' to facial stimuli for the placebo and alcohol groups (maximum frequency = 90).

		emotion responses					
PLACEBO		**hap**	**sad**	**fear**	**ang**	**surp**	**disg**
stimuli	**happiness**	90	–	–	–	–	–
	sadness	–	84	4**	–	1	1
	fear	–	1	72	3	10**	4
	anger	–	2	–	71	3	14**
	surprise	4**	1	8**	1	76	–
	disgust	2	–	–	14***	–	74
ALCOHOL		**hap**	**sad**	**fear**	**ang**	**surp**	**disg**
stimuli	**happiness**	90	–	–	–	–	–
	sadness	–	74	2	–	5	9*
	fear	–	2	48	5	27**	8
	anger	–	4	1	72	5	8
	surprise	1	1	6***	–	80	2
	disgust	1	1	–	16***	2	70

* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

Table 8.8. Frequency of 'emotion response' to oral stimuli for the placebo and alcohol groups (maximum frequency = 60).

		emotion responses					
PLACEBO		**hap**	**sad**	**fear**	**ang**	**surp**	**disg**
stimuli	**happiness**	43	6	2	3	5*	--
	sadness	--	54	6**	--	--	--
	fear	--	16***	39	2	3	--
	anger	--	--	1	51	2	6*
	surprise	6***	4	11*	1	36	--
	disgust	--	--	--	22*	1	37
ALCOHOL		**hap**	**sad**	**fear**	**ang**	**surp**	**disg**
stimuli	**happiness**	41	3	6	1	6	1
	sadness	--	54	4	--	--	2
	fear	--	15**	39	--	6	--
	anger	--	1	2	50	2	5***
	surprise	3**	4	7	--	45	--
	disgust	1	1	--	19***	--	39

* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

Separate 2x2 ANOVAs were performed for each emotion in order to explore the influence of alcohol intoxication and medium of expression on the accuracy to recognize emotional expressions of the different emotions. Results of the six ANOVAs are shown in table 8.6.. The main effect of medium was statistically significant for three emotions: happiness, fear and disgust. It was more difficult to recognize these emotions when expressed orally (see table 8.5.). The subjects who consumed alcohol tended to recognize expressions of fear less accurately, which is reflected in an almost significant main effect of group ($F(1,28)=3.69$, $p=0.064$). There was also a statistically significant interaction effect on this emotion ($F(1,28)= 4.33$, $p=0.046$). Analysis of simple effects showed that alcohol consumption did not affect recognition of oral expression of fear ($F<1$), whereas it did reduce accuracy in the recognition of fear expressed in the face ($F(1,28)=13.13$, $P=0.001$). In the present study, no effect of alcohol was found on judgement of facial expressions of anger or disgust. Only on sadness was a slight trend found: intoxicated subjects tended to perform worse in the recognition of facial expression of sadness ($F(1,28)=2.56$, $p=0.1$). There was a trend for more accurate recognition of surprise expressions when intoxicated ($F(1,28)=3.18$, $p=0.085$). Simple analysis revealed that alcohol tended to improve only judgement of oral expressions (for facial material $F<1$; for oral stimuli $F(1,28)=2.97$, $p=0.095$).

Recognition of facial identity

The placebo group scored slightly higher on the Benton Test of facial

recognition, although this difference was not statistically significant (alcohol group: mean= 20.40, sd=2.03; placebo group: mean=21.53, sd=2.42 ; t=1.31, df=27.19, p= 0.202).

In order to investigate the relationship between scores on Benton's Test and performance on the judgement of facial and oral expressions of emotion, product-moment correlation coefficients between scores on Benton's test and the total number of correct categorization of facial and oral emotional expressions were calculated.

Whereas a positive statistically significant correlation was found between performance on the Benton Test of facial recognition and the number of correct categorizations of facial expressions of emotion (r=0.379, n=30, p=0.038), there was no correlation between Benton's test and recognition of emotion expressed in the voice (r=0.133, n=30, p=0.482).

Confusion matrices

Tables 8.7. and 8.8. show respectively the confusion matrices for judgement of facial and oral expressions of emotion, computated separately for each treatment group. The equiprobability model (that is, whether the recognition errors are distributed at random) was tested on each of the four matrices separately by means of the procedure 'Loglinear' of the SPSS statistical package (Norusis, 1990). This program allows to define as structural zeros the cells representing the correct recognitions - the principal diagonal of the matrix. In tables 8.7. and 8.8. the cells which deviate significantly

from the equiprobability model have been marked. The p values are based on the standardized residuals of each cell. The placebo and alcohol matrices were compared for each medium (oral and facial) separately. T-tests were used to compare those cells which both in the alcohol and in the placebo matrices deviate significantly from the equiprobability model. For the facial medium only one contrast was statically significant. Those who drank alcohol mistook faces of fear for surprise more frequently than those who did not ($t=2.83$, $df=24$, $p<0.01$). No test was statistically significant in the oral medium.

8.4. Discussion

The placebo manipulation in this experiment worked satisfactorily, since subjects in the placebo group believed they had drunk alcohol. However, it seems that, as they could not feel any strong effects of intoxication, they estimated that they had drunk a small amount. The dose of alcohol the placebo subjects guessed they had received was lower than that of the alcohol group, although the amount of alcohol they estimated was quite substantial: an average of 1.9 pints of beer. The experimenter observed that some subjects in the placebo group, when asked to estimate the alcohol they had drunk, gave answers such as the following, "I do not think I have drunk much. I do not feel drunk. At the beginning may be, but now I feel OK", and proceeded to estimate that they had drunk the equivalent of one or two pints of beer. One could predict that the consumption of this amount of alcohol in 18 min on an empty stomach would lead to a substantially higher degree of intoxication. One might speculate that people do not drink with their stomachs empty so often; that when

they drink they do it in an atmosphere that does not encourage the scanning of own sensations; and that these and other factors may account for their reports. In sum, the analysis of the placebo manipulation in this experiment highlights the difficulty of controlling all the nonpharmacological factors by means of placebo procedures when the drug under scrutiny is ethanol.

Stress was reduced equally in both groups. It is impossible to ascertain whether this was an effect of the belief of having drunk alcohol or the result of habituation or familiarization with the environment.

Arousal 30 min after consumption was clearly increased by alcohol, although this arousing effect did not last long. These results could be interpreted as an arousing effect of alcohol confined to the ascending limb of the BAC curve.

Alcohol seems to influence the categorization of only facial expressions as revealed in the alcohol emotion interaction effect, which was statistically significant only for facial expressions. In table 8.3. it can be observed that those who drank alcohol used the 'fear' label less and the 'surprise' category more than those who received a placebo. Analysis of variance on fear and surprise responses to facial expression stimuli showed that these differences were statistically significant.

While in the recognition of emotional facial expressions in the placebo group the frequency of use of the different labels did not vary much, subjects in both groups used the sad and anger labels more frequently

than others when categorizing vocal expressions of emotion. Was this due to a tendency of amateur actors to produce utterances that are more readily interpreted as sadness or anger? That is a question that remains to be answered. However, as far as the aim of the present study is concerned, the issue is that judgement reponses to expressive oral material did not differ between alcohol and placebo groups.

While alcohol did not affect the accuracy of judgement of oral expressions at all, there was a tendency for those who did not drink alcohol to perform better in the labelling of facial expressions, although this effect did not reach statistical significance ($p=0.089$). It should be noted, nevertheless, that the size of this effect is comparable to the one found by Tucker and Vuchinich (1984), since they found that the impairment caused by alcohol was statistically significant only when using a one-tailed test. The effect found by Borrill et al. (1987) was greater: although the pairwise comparison using Scheffe's method between placebo and high dose subjects was not significant, examination of their means and standard deviations reveals that the difference is statistically significant if a less conservative test (e.g. a t-test) is used. Taken together, the results of these studies (plus the one by O'Sullivan et al.,1975, from which we only have indirect information) and the present one suggest that alcohol produces a deleterious effect on the judgement of pictures of facial expressions of emotion, which is replicable, but of small magnitude (this is why it does not always emerge as statistically significant).

The analysis of accuracy in the judgement of facial expressions of the different emotions reveals that alcohol dramatically reduced the

accuracy of perception of fear expressions. The analysis of the confusion matrices reveals that most misjudged expressions of fear were taken as surprise. This tendency for alcohol to produce confusion of fear and surprise was statistically significant. The confusion of fear for surprise is one of the most likely to happen. In a recent study (Ekman et al, 1987), subjects of 10 different cultures were presented a series of facial expressions (the same material used here) twice. The first time they were asked to recognize the emotion; the second time they were told that expressions might show more than one emotion and asked to rate each of the emotions in terms of whether they were present or absent in the face, and in what strength. In the ten cultures the secondary choice for all the expressions of fear that were shown was surprise. If alcohol exerts an impairing effect on visual perception this would exacerbate this confusion, which is exactly what happened in the present study.

Although it did not reach statistical significance, there was a trend for subjects who consumed alcohol to use the surprise label more frequently. This tendency is accompanied by (and may be causing) the higher accuracy in the judgement of oral expressions of surprise in the alcohol group. Although one must bear in mind that these effects did not reach statistical significance, it seems as if alcohol led to the recognition of surprise at the expense of fear on both media, which would suggest an effect of alcohol of an emotional rather than perceptual nature. However, a cognitive explanation seems more parsimonious. The confusion of fear for surprise was more marked and statistically significant only in the facial medium, where the recognition of the expressions of these emotions are particularly prone to confusion. Moreover, the positive correlation between scores on

Benton's test and correct identifications of facial expressions definitively supports the cognitive hypothesis.

The cognitive explanation is supported also by the fact that, although a small detrimental effect of alcohol on the recognition of facial emotion has been a consistent finding, the intensity of the effect of alcohol on the recognition of the different emotions has been different in each study. O'Sullivan et al (1975) found that alcohol particularly impaired the recognition of anger and sadness. Borrill et al (1987) found the largest effect of alcohol on the recognition of anger and disgust. In the present study alcohol made subjects mistake particularly fear for surprise. It seems that the effect of alcohol consists in exacerbating the confusions between emotions which are perceptually more difficult to distinguish.

What is the relevance of this cognitive impairment in real life social interactions? One could speculate that, with information from the face becoming less reliable, contextual information might become more influential in determining the judgement of others' emotions during social interactions. Clarity of each source of information (face and context) has been pointed out as one of the parameters that determines the relative importance of judgements of emotion (Ekman, Friesen and Ellsworth, 1982a). Context as we understand it includes environmental cues, bodily feedback, expectancies, etc. When facial cues are less distinctive and discriminable, learned expectancies about the effects of drinking and relaxing effects of alcohol become more important.

Chapter 9
GENERAL DISCUSSION

Alcohol and sexual disinhibition

In a recent review the possible pharmacological disinhibitory effects of alcohol on sexual response were judged as unimportant in relation to the influence of other factors such as 'cognitive variables', 'expectancies', etc. (Roehrich and Kinder, 1991). However, the experiment reported in chapter 3, which replicated and extended the results of the study by Wilson and Niaura (1984), showed that alcohol can effectively disinhibit sexual response if the individual is trying to suppress his response to erotic stimuli voluntarily. The traditional view of alcohol as an aphrodisiac substance might have some basis. Recently Pfaus and Pinel (1989) found that alcohol had a disinhibiting effect on sexual behaviour of male rats only if the sexual response had previously been inhibited. In a first experiment Pfaus and Pinel (1989) found, as expected, that all doses of a range from 0.25 to 2.0 g/k reduced copulatory behaviour in male rats. In a second experiment the male rats were subjected to a training phase in which they repeatedly encountered nonreceptive females. This training resulted in inhibition of the copulatory behaviour of the males. After a dose of 0.5 g/k of ethanol most of the males previously made nonresponsive by the inhibitory training mounted the nonreceptive females. A higher dose of 1.0 g/k did not have this inhibitory effect. Pfaus and Pinel (1989) opine that their results fit well in the theory proposed by Steele and Southwick (1985). According to this theory, when behaviour is controlled by both inhibiting and instigating pressures, the cognitive impairment caused by alcohol will impair the processing of the

inhibitory cues, and, as a consequence of this, behaviour will be exclusively determined by the instigating cues. This theory necessarily requires the assumption that the inhibitory cues are less salient or more difficult to process that the instigating stimuli. Steele and Southwick (1985) understand that the inhibitory control, that alcohol blocks, consists, at least in humans, in some sort of "inhibitory thoughts" (p. 21) which come after the instigation. The results of the experiment in chapter 3 suggest that alcohol impaired the processes by means of which subjects tried to control (i.e. inhibit) the ongoing sexual response. It is difficult to determine to what extent the phenomenon found by Pfaus and Pinel (1989) and the one described in chapter 3 are comparable. Steele and Southwick (1985) could represent a common theoretical framework to understand both phenomena only at the expense of adopting the most comprehensive and laxest version of Steele and Southwick's (1985) theory, according to which the inhibitory processes are some kind of unspecified higher mechanisms which are more easily impaired by alcohol than the mechanisms underlying the instigation of sexual response. The echo of Jackson's theory (chapter 1) resounds in this formulation.

Animal data have usually (e.g. Wilson, 1981) been disregarded as irrelevant to understand human sexual response and the effects of alcohol on it. However, recently, the study of sexual response in laboratory rats has produced several behavioural paradigms that allow one to study a number of motivational aspects of sexual behaviour besides the consummatory behaviours of mounting and copulating (see Everitt, 1990). These conceptual and methodological developments have served to study the role of different neurotransmitter systems and different structures of the CNS in the different aspects of sexual

behaviour. It has been suggested that these developments legitimate the use of rodents' data in the effort to understand human sexual response (Everitt and Bancroft, 1991). Although extrapolation from animal data should always be taken with caution, the subtle analysis that these paradigms have made possible, for example, of the role of the dopaminergic system in the different aspects of the sexual response fully justifies that some effort is put into the investigation of the effects of alcohol on sexual response using these new paradigms. The study by Pfaus and Pinel (1989) shows that animal data can be relevant.

It is obvious that it is impossible to study the different aspects of human sexual behaviour with the degree of control that rat's behaviour is investigated. The paradigm used to study experimentally human sexual response has consisted in measuring the subjective and phychophysiological sexual response to different kinds of sexual stimuli (i.e. still visual stimuli, erotic movies, erotic narratives, fantasy, etc.). Using this experimental paradigm it has been shown that the erectile response to erotic fantasies is androgen dependent while the response to visual (filmed) erotic stimuli is not (see O'Carroll, 1988). On the basis of these findings (together with the observed dependency of sexual desire and spontaneous nocturnal erections on androgens) it has been hypothesized the existence of two systems in the brain: one androgen dependent and other which is not influenced directly by androgen hormones (Bancroft, 1988). This experimental paradigm has served to study human sexual response under different conditions of induced anxiety and performance demand manipulation, which has made it possible to outline differential characteristics of functional and dysfunctional individuals (see Cranston-Cuebas and Barlow, 1990). These are mentioned here as examples of the benefits that the use of

this experimental paradigm has produced. (Amongst these benefits we should also count the results of the investigation on the effects of alcohol on sexual response reviewed in chapter 2.) However, the present techniques are not fully satisfactory. The manipulation of sexual stimuli or information can be said to be somewhat crude. We need a better understanding of how sexual information is processed. Various authors (e.g. Geer and McGlone, 1990, Cranston-Cuebas and Barlow, 1990) have proposed that sex research has much to gain by borrowing from cognitive psychology techniques. Both Barlow and Geer agree that the techniques Mathews and his associates (e.g. Mathews and MacLeod, 1986, Mathews et al , 1990) have been using to study processes such as attention allocation, perceptual defence and mood congruent memory in anxious individuals might be useful in understanding information processing of sexual stimuli. It has been suggested that alcohol-induced cognitive impairment might explain the reduced ability to suppress ongoing sexual response. When debriefed, the subjects in the experiment in chapter 3 were invited to describe how they tried to suppress their response. The usual method reported was to try to think of something else or not to focus the attention on the erotic stimuli. It seems therefore that erotic stimuli attracted attention in a more powerful way when the subject had drunk. This possibility and other possible aspects of the processing of sexual information should be investigated. It might be argued that to say that alcohol impairs cognitive processing is a too vague and unsatisfactory explanation. The disruption of cognitive processes has also been proposed to explain the reduction in sexual response found in a number of studies (Wilson et al, 1985, Bancroft, 1989). It is necessary to identify what cognitive processes are influenced by alcohol and in which way these effects can account for the effects of alcohol on sexual response. This highlights the

importance of understanding the processing of sexual information. In any case, the finding that alcohol impairs the voluntary suppression of sexual response in males is a significant phenomenon no matter what processes mediate this effect.

Alcohol and perception of expression of emotion

The experiment reported in chapter 8 which examined the effects of alcohol on perception of emotional expression also suggests an effect of alcohol of mainly cognitive nature. In this experiment the so-called categorial paradigm was used. This paradigm, in which the individual is presented with an example of emotional expression and asked to select one category, provides, as Dittman (1973) has explained, information about the subject's competence to discriminate and classify emotional stimuli "under ideal circumstances". The dimensional approach in which the subject is asked to rate each stimulus on several scales (the most frequently used scales being activation - passive-active scale - and pleasantness) might "tell us more about how the person approaches an expression and what sorts of things he pays attention to first" (Dittman, 1972, p.73). The dimensional approach could provide complementary information to that obtained from the categorial paradigm. Future research should examine the effects of alcohol on perception of emotional expression by using the dimensional paradigm. On the other hand, research should be carried out in order to determine the characteristics of the impairment in visual perception that seems to underlie the deficit in perception of facial expression of emotion that alcohol causes. Robertson et al (1985) have found that alcoholics present difficulties in perceiving configurations of elements, and this impairment was not caused by tunnel vision since the distance

between elements did not influence the deficit of alcoholics. It should be worth exploring whether alcohol intoxication produces the same kind of deficit and whether it mediates the impaired perception of emotional facial expressions.

Alcohol and the response to social stress

The reduction in the stress-caused heart rate increase is one of the most consistent effects of ethanol (see chapter 4). It was found in chapter 6 that the self-disclosure task, contrary to what has been claimed (Abrams, 1983), strongly habituated. Despite this problem the repeated measures design reported in chapter 6 provided some evidence that the effects of alcohol on HR also occur at doses below 0.8 g/k. Further research should investigate the possibility of developing non-habituating anxiety-producing tasks in order to be able to profit from the advantages of the cross-over design.

Psychological experiments very often involve an interaction between subject and experimenter that must be understood as a social relationship. If this is true of experiments examining performance or pure intellectual processes, the importance of this relationship cannot be overemphasized in those studies in which the experimenter is himself an important part of the stimulus situation. The experiment in chapter 7 has shown that experimenter's behaviour should be treated, particularly in studies involving social interaction with the subject, as an outcome variable that can be influenced by the experimental factors and the process of conducting the experiment, and that should be measured and analyzed. The way the experiment was conducted is equivalent to the not uncommon practice of testing the control group

first and then the group of alcoholics.

Alcohol and the startle/defence response

I have argued for the need to break down molar behaviour such as the response in the social stress task into simpler components in order to examine the effects of alcohol on more basic processes. Chapter 7 represents an attempt to examine the effects of alcohol on a primary response to stimulation (that of intake/rejection). The results of this experiment suggest that alcohol made the subject react to stimulation in a less receptive way. The results of this experiment, however, should be viewed with some caution due to the differences in pre-treatment anxiety. This study was conceived as a tentative endeavour, in that it was the application of a theoretical framework not previously used to study alcohol intoxication and in that it involved the development of a technically complex experimental setup that was tested in this experiment. From a theoretical point of view, I argued that the startle/defence reflex indicated a response of emotional nature, against some authors' view (e.g.Ekman, 1984) that the startle response was a mere reflex. Several studies published after the completion (or at least the commencement) of the experiment reported here have confirmed the emotional nature of the startle reflex and its utility to study the emotional state of the individual. Vrana et al (1988) found that the SR evoked by auditory stimuli was larger when subjects were viewing aversive slides (e.g. mutilated bodies) than when they were contemplating slides depicting pleasant things (e.g. attractive models, happy infants). The issue of the nature, whether emotional of attentional, of the startle response has been addressed in a recent experiment by Bradley et al (1990). They found that SR amplitude

varied as a function of the emotional valence (neutral, pleasant or unpleasant) of the slides viewed by the subjects when the startle probe (auditory or visual) was presented and was independent of the modality-determined attention allocation, confirming therefore the emotional nature of the SR. Cook et al (1991) also found that the startle response was potentiated in imagery-evoked negative affective states. The conception of startle reflex in these studies is as a "protective or defensive response" to aversive stimuli. During the last few years such work has demonstrated that this defensive reaction is enhanced in states of negative affect. The dependent variable used in these studies has been the blink reflex. It has been concluded that these studies "demonstrate a practical procedure for assessing emotional (particularly fear) states. As the eyeblink reflex is obligatory, and probes are presented unpredictably, volitional influence on the response is minimal. (...) Thus, this methodology has much to recommend it as a tool for emotional assessment in basic research of emotion, emotional development, and the psychopathology of affect." (Vrana and Lang, 1990). This paradigm should be an useful instrument to study the effects of alcohol on emotional state. An interesting aspect of this paradigm is that it could be repeated during the same session at different points of the blood alcohol concentration curve, and on different occasions in a cross-over design.

Pharmacological vs. psychological causation of post-drinking emotional behaviour

Sociology of science has pointed out what has been called 'interpretative flexibility' of all scientific evidence (Collins, 1981). That is, scientific data never lead to a unique and straight forward

interpretation (even when the scientist working within a particular tradition or framework sees only one obvious interpretation). The so-called 'closure of controversy' process results in the imposition of restrictions in the flexibility of interpretation by defining what is going to be seen as the only plausible interpretation ('monopolisation of plausibility'). Mc Ancrew and Edgerton's (1969) collected numerous examples of anthropological evidence to show that the effects of alcohol varied widely amongst cultures and social groups. They concluded that people learn what to expect from alcohol from their society and that these socially learned expectancies, and not the pharmacological action of ethanol, determine the actual consequences of alcohol intoxication. Mc Andrew and Edgerton's (1969) work set up the scene for the interpretation of BPD studies in the way it was done, for example, by Marlatt and Rohsenow (1980) - see chapter 1. In terms of the theory of 'closure of controversy', the papers by MacAndrew and Edgerton (1969) and Marlatt and Rohsenow (1980) 'closed the controversy' about the origin of the behavioural effects of alcohol intoxication by emphasizing the predominant role of psychological factors. The pharmacological action of alcohol was disregarded and any explanation invoking psychological mechanisms sounded plausible. However, one might argue that it is one thing to show that psychological factors can influence what we experience and how we behave after consumption but it is another to conclude that ethanol is an inert substance or a drug whose effects are behaviorally irrelevant. The way a culture teaches the individual what to expect from alcohol and how it coaches him or her to deal with drinking and alcohol intoxication will, without doubt, interact with the pharmacological action of ethanol, even antagonizing its effects. But in most situations the alcohol will be a major agent determining the behavioural consequences of drinking. It

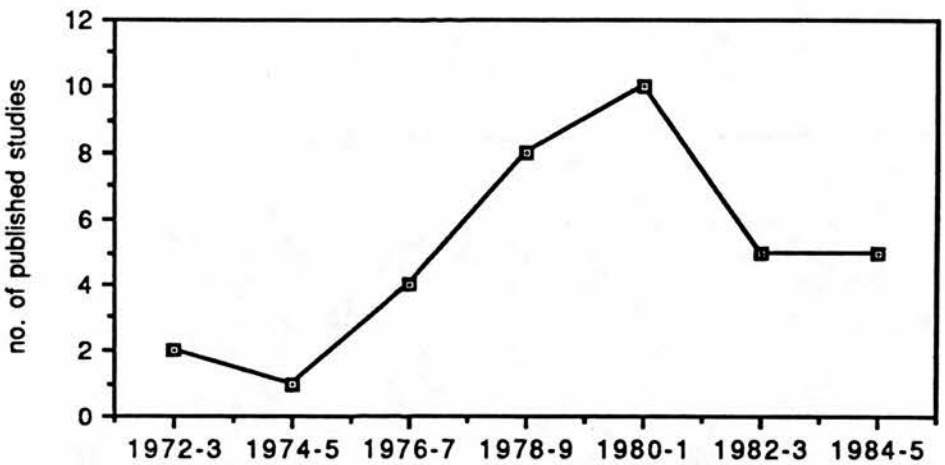

Fig. 9.1. - Number of BPD studies published. Only studies included in the Hull and Bond (1986) review are included.

also conceivable that cultural beliefs about alcohol reflect to a certain extent the real effects of ethanol. However, we cannot expect to find descriptions of alcohol intoxication in terms of scientific psychological theory in the folk wisdom about alcohol, but constructions in terms of the models of behaviour of that culture.

The emphasis on the psychogenic determination of the effects of alcohol intoxication has receded and after a peak circa the turn of the decade the number of BPD studies published has declined in the 1980's (see Fig. 9.1.). One of the reasons for this decline is, without doubt, methodological, and I shall deal with it below. Another reason is that the emphasis has shifted towards the recognition that alcohol has significant effects on the organism and it is the main responsible for the effects of alcohol intoxication (e.g. Hull, 1987, Levenson, 1987, Sher, 1987, Steele and Southwick, 1985), and consequently researchers are more concerned with demonstrating the pharmacological effects of ethanol than the influence of nonpharcological, psychogenic factors.

The so-called cognitive explanation of the pharmacological effects of alcohol on emotion

Most of the new theories on alcohol intoxication "assume that some of the causes and effects of alcohol consumption can be understood in terms of the pharmacologic effects of the drug" and propose that alcohol affects behaviour indirectly by virtue of its effects on cognition" (Hull, 1987, p. 272). With some exceptions such as Levenson (1987) who thinks (very likely not without justification) that alcohol makes you feel 'good' ("the thrust of our argument compels us to propose that the visceral state in which we find our intoxicated subjects is a desirable

one", p. 180), the emphasis is presently on the cognitive effects of alcohol. It is believed that "alcohol does have direct reliable effects, but not on mood or emotion. Rather, alcohol intoxication directly affects information processing" (Josephs and Steele, 1990, p. 125).

The results of the experiments presented here have also supported the cognitive explanation of the effects of alcohol intoxication on emotion. Let us examine some aspects of the cognitive models of the emotional effects of alcohol consumption. Firstly, as Steele and Southwick (1985) have warned, the demonstration that alcohol influences cognitive processes which might mediate the emotional response does not represent direct evidence that the social and emotional effects of alcohol result from impairment of cognitive processing. A good example of this problem is the study by Yankofsky et al (1986) reviewed in chapter 8. Yankofsky et al (1986) intended to examine the effects of alcohol on perception of interpersonal feedback and self-evaluation in the assumption that these processes determine the anxiety response. Although an effect of alcohol on perception of negative feedback was found, alcohol did not have a significant effect on the anxiety response.

The basic assumption in all cognitive theories of alcohol intoxication is that alcohol disrupts "higher-order cognitive processes" and that disinhibition and reduced anxiety derives from this disruption. Behind this hypothesis there is the assumption that inhibition and anxiety depend always on higher cognitive processes. On reading Hull (1981, 1987) or Steele (Steele and Southwick, 1985, Josephs and Steele, 1990) it is impossible not to be reminded of the classic formulations of the disinhibitory theory by Jackson or Schmiedeberg. Hull (1987) has proposed that alcohol reduces anxiety "by inhibiting higher-order

cognitive processes relating to the encoding of information in terms of its self-relevance" (p. 275), and for Hull the encoding of information as self-relevant underlies self-conciousness. Cushny (1899), a disciple of Schmiedeberg, explained the view that alcohol produces "the weakening of the highest functions of the brain -the will and self-restraint" (p. 133). One may argue, however, that emotional response, and in particular anxiety, does not necessarily depend on higher-order processing. Moreover, it can be argued that at least some aspects of anxiety are a primary response to stimulation.

As it was argued in chapter 1, the emotional response is best understood in terms of a cycle of environment-individual exchanges, i.e. the emotional cycle. We should study how alcohol affects the different aspects of this cycle.

When confronted with a stimulus the individual might respond either in a receptive or in a defensive way. Following Arnold (1960) and Fisher et al (1990) amongst others, I proposed in chapter 1 that the primary response to stimulation consisted in either a positive (approaching, receptive) or a negative (withdrawal, rejection) reaction to the stimulus. This conception exactly coincides with Lang's model of emotional responding in the light of which the results of the studies using the startle reflex probe paradigm carried out by him and his associates in the last few years have been explained: "The organism is efferent tuned by context and internal state, either appetitively (represented primitively as approach, attachement, and consumatory behaviour) or defensively (as avoidance, escape, deffensive aggression)." (Bradley, Cuthbert and Lang, 1990). Alcohol might affect this primary emotional response, and, as proposed above the startle response

paradigm seems the appropiate technique to investigate the effects of ethanol on this aspect of the emotional cycle.

Limited capacity of processing due to alcohol intoxication in certain circumstances, i.e. when distractors are present, might lead to a 'neglect' of "inhibitory thoughts" and a reduced processing of anxiogenic stimuli in anticipation of a stressing situation (Josephs and Steele, 1990). A reduced cardiac response to stressing stimuli has been consistently found. Whether this is a direct effect of alcohol on cardiac response or the indication of a dampened emotional reaction has not been totally clarified yet. Beta-adrenergic blockers that reduce cardiac activation have a limited effect on anxiety: they relieve anxiety only in those cases in which the somatic symptoms are predominant, but are ineffective in those cases in which the subjective component is important (Lader, 1982). However, the combination of reduced cardiac activation and decreased higher processing of anxiogenic cues might have effective anxiolytic benefits.

It has been explicitly proposed that the effects of alcohol on emotion are an indirect result of the influence of alcohol on cognition or information processing (e.g. Hull, 1987, Josephs and Steele, 1990, Yankofsky et al, 1986). When considering the relationship between cognition and emotion one faces the conceptual problem that the notion of cognition has been developed to mean those processes exempt of emotional connotations, and the concept of emotion to indicate that type of processes in the organism which are not cognitive. This absolute separation of cognition and emotion is not satisfactory. The close imbrication of emotional and cognitive aspects in all psychological processes is today widely recognized. The information processing

approach to the study of emotion adopted here (chapter 1) rests on this idea. The basic intake/rejection response to stimulation is a good example of the impossibility of separating cognition and emotion if cognition is understood as information processing.

As BPD studies have shown, psychological factors can be important determinants of post-drinking behaviour. This can be done in different ways. For example, learned expectancies could influence the affective state of the individual, which will influence the response to the startle response. Later in the emotional cycle the influence of expectancies could be even more important, determining the secondary processing of the emotional situation and the regulatory mechanisms to be deployed (see chapter 1).

The problem of the design

The BPD was developed in order to control and to study the influence of nonpharmacological factors. Several studies have analyzed the works and limitations of BPD. Bradlyn et al (1983) found that many of the subjects, in a BPD, who were told that their drinks did not contain alcohol but were given a dose of 0.69 g/kg, began to suspect the deception shortly after they had finished their drinks. Knight et al (1986) investigated the influence of demand characteristics on subjects' answers to the questions posed to them in the debriefing. Knight et al (1986) first conducted the manipulation check in the usual way. Then, they told the subjects that the file containing their group allocations had been lost due to a computer breakdown. A substantial number of subjects (36% in the group receiving a soft drink while being told that their drinks contained alcohol, and 90% in the group who was told that

their drinks did not have alcohol although they actually did) changed their responses, revealing that they suspected the deception. The manipulation checks conducted in chapter 3 confirmed the problems of BPD. It was found that those who actually received alcohol estimated to have consumed a significantly larger amount of alcohol than those who did not receive alcohol. Despite these problems the BPD has made a valuable contribution to our understanding of alcohol intoxication. Although subjects might have suspected the deception more often than recognized, in many experiments the experimental instructions might have been effective as subjects' beliefs about what they have drunk effectively determined behaviour. However, the limitations of the BPD seem clear: with doses above 0.6 g/kg and more than 30 min after consumption subjects can be expected to suspect the deception.

It has been argued (Cappell and Greely, 1987) that research on alcohol effects should adopt simpler experimental designs in order to best ascertain the real effects of alcohol. A simpler design, in which two groups (placebo and alcohol) are told that they are getting alcohol although only the alcohol group gets it, has been used in two of the experiments in this thesis. This simple design is not free of problems, though. Although subjects in the placebo group did not show any sign of suspecting the deception they felt less intoxicated and estimated to have drunk a lower amount of alcohol.

The psychopharmacology of alcohol faces the problem that practically all subjects are able to recognize the sensations of alcohol intoxication. This makes it practically impossible to isolate the pharmacological effects of alcohol in experiments in which subjects know alcohol is involved. If we want to ascertain its real effects, alcohol will have to be

administered in an experimental context in which subjects are not told that alcohol is given. They could be informed, for example, that the aim of the study is to compare the effects of different anxiolytic drugs.

It has been commonplace for last few years in this field to say that the research question is not whether alcohol reduces anxiety or increases sexual arousal, etc., but in which subjects, in which circumstances, etc. The proposal being made here is that the research in this field should aim to study the effects of alcohol on the basic processes of emotion in experiments in which subjects cannot suspect that the drug being tested is alcohol.

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APPENDIX I

Information sheets, consent forms, questionnaires and visual analogue scales used in the experiments.

UNIVERSITY OF EDINBURGH

Department of Psychology

Study: ALCOHOL AND SEXUAL AROUSAL

I understand that my erectile response and my heart rate will be measured while I watch an extract from an erotic film, which contains actual human intercourse and oral sex. The techniques used to take these measures have been fully explained to me. I am aware that half of the subjects taking part in the experiment will have a moderate dose of alcohol. I understand that I am free to withdraw from the experiment at any stage.

Signed.....

Date .....

UNIVERSITY OF EDINBURGH

Department of Psychology

Study: ALCOHOL AND SEXUAL AROUSAL

(Information to subjects participating
in the experimental study)

The present study aims to examine the effects of alcohol on some aspects of sexual response to erotic stimulation -erotic films containing actual human intercourse and oral sex.

The experiment involves two groups: an experimental group and a control group. The erectile response of the penis, the heart rate as well as the subjective arousal of each subject will be measured while watching an erotic video. A moderate dose of alcohol will be given to those subjects allocated to the experimental group. The assignation to each of the groups will be random and will be carried out at the start of the experimental session. Every subject in the experiment must be prepared to receive a moderate dose of alcohol.

As usual in experiments of this nature, all factors except the dose of alcohol taken must be kept constant across groups. Hence, all of the subjects are asked to accomplish all requirements listed below. As well, subjects in both groups will consume the same volume of liquid and at the same rate of intake. All this isolates those effects which are due to the action of alcohol alone.

The requirements that all of the subjects are asked to meet before the experimental session are:

1. not to have eaten for 4 hours
2. not to have had sexual activity for at least 12 hours
3. not to have drunk alcohol for 12 hours
4. not to have had any drug or medicine for 12 hours

Breath alcohol concentration will be measured at the start and at several points during the session.

We must make clear to all of the subjects taking part in this study that sexual arousal experienced in such an artificial setting might be quite different from normal reactions in real life.

In this study we are not interested in the response of each particular person but in the differences between experimental and control groups generally.

If you have failed to keep any of the requirements, do not hesitate to tell the experimenter. He will weigh up the importance of the failing and will give you an appointment for another day if your omission has been important enough.

COMMENT ABOUT THIS KIND OF RESEARCH

There is evidence coming from different areas of research that drinking generally enhances human sexual arousal. The personal experience of most people confirms the 'academic' evidence. However, to understand how alcohol influences or controls our sexual response we have to take very accurate measures of both subjective (what you feel) and physiological (what the scientist measures) sexual response in a way that can only be carried out in the controlled environment of the laboratory.

If we compare the response to erotic material (the same material for all the subjects) of a group of people who have drunk with that of another group who have not drunk alcohol, we will be able to get some

useful information about the action of alcohol. When sexual response is measured very precisely as in this experiment, information can be obtained about the underlying psychological and/or physiological processes and mechanisms involved. Nevertheless,, the aspects of and the scale of response in which we are interested are generally irrelevant in real life.

PROCEDURE TO MEASURE ERECTILE RESPONSE

You are probably concerned about your erectile response being recorded in the experiment. You probably think that it will be embarrassing for you. However, the procedure that will be used assures complete privacy. The erectile response is recorded without putting the subject in an embarrassing situation.

The device that will be used consists of a tiny rubber ring which is placed around the penis. This ring consists of a thin silicone tube (0.082 in.) filled with mercury. The electrical resistance of the mercury varies according to the diameter of the ring.

It is important to make clear that you will place the penile device in complete privacy. Once the device has been placed it can be worn under the clothes and you will probably forget that it is there. During the experiment you will stay alone in a room comfortably seated.

Before the experiment, you will be shown the penile device and other intruments to be used; the procedure of measurement will be fully explained to you. Afterwards you will be able to consider your participation. If you decide to take part in the experiment you will still be free to withdraw at any stage.

We honestly think that your participation in the experiment will not lead to an unpleasant experience. However, we understand that any personal attitudes or decisions must be respected.

Subject no.: .....

This questionnaire is completely confidential.

Please ring the appropiate number or write down the answer when that is required.

1. Have you ever suffered from any liver problem?

1. yes 2. no

2. Have you ever suffered from stomach or duodenal ulcers?

1. yes 2. no

3. Are you currently taking any medication or drug (prescribed either by a doctor or by yourself)?

1. yes 2. no

If yes, specify .....

4. Have you ever been advised not to drink alcohol because of any health problem?

1. yes 2. no

If yes, why? .....

5. Do you have any current health problems?

1. yes 2. no

If yes, what? .....

6. Have you ever consulted a doctor, psychiatrist or psychologist about any sexual problem?

1. yes 2. no

If yes, for what type of problem did you seek advice? .....

.....

Subject no.: _____

How do you feel? Please indicate by marking the line in the appropiate place according to the degree to which you are experiencing each feeling listed.

	NOT AT ALL	EXTREMELY
1. Sexually aroused	0	10
2. Liked	0	10
3. Offended	0	10
4. Romantic	0	10
5. Bored	0	10
6. Disgusted	0	10
7. Uncomfortable	0	10
8. Entertained	0	10
9. Interested	0	10
10. Intoxicated	0	10
11. Embarrassed	0	10
12. Guilty	0	10
13. Anxious	0	10
14. Ashamed	0	10
15. Relaxed	0	10
16. Uneasy	0	10
17. Afraid	0	10
18. Drunk	0	10
19. Nervous	0	10
20. Carefree	0	10
21. Excited	0	10
22. Sleepy	0	10

Subject no.: .....

ow SEXUALLY AROUSED did you feel while watching the video? Please indicate
y marking the line in the appropiate place between the two extreme options.

OT AT ALL
ROUSED 0 ——————————————————— 10 EXTREMELY AROUSED

Subject no.: .....

What degree of erection did you reach while watching the erotic video?

ERECTED
LL 0 ——————————————————— 10 FULL ERECTION

Subject no.: .....

' SEXUALLY AROUSED did you feel for this time while trying to get aroused?
ase indicate by marking the line in the appropiate place between the two
reme options.

' AT ALL
USED 0 ——————————————————— 10 EXTREMELY AROUSED

Subject no.: .....

What degree of erection did you reach while trying to get aroused?

RECTED
L 0 ——————————————————— 10 FULL ERECTION

SELF-EVALUATION QUESTIONNAIRE

STAI FORM X-2

Subject no. ..... Session ..... Phase .....

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
21. I feel pleasant	①	②	③	④
22. I tire quickly	①	②	③	④
23. I feel like crying	①	②	③	④
24. I wish I could be as happy as others seem to be	①	②	③	④
25. I am losing out on things because I can't make up my mind soon enough	①	②	③	④
26. I feel rested	①	②	③	④
27. I am "calm, cool, and collected"	①	②	③	④
28. I feel that difficulties are piling up so that I cannot overcome them	①	②	③	④
29. I worry too much over something that really doesn't matter	①	②	③	④
30. I am happy	①	②	③	④
31. I am inclined to take things hard	①	②	③	④
32. I lack self-confidence	①	②	③	④
33. I feel secure	①	②	③	④
34. I try to avoid facing a crisis or difficulty	①	②	③	④
35. I feel blue	①	②	③	④
36. I am content	①	②	③	④
37. Some unimportant thought runs through my mind and bothers me	①	②	③	④
38. I take disappointments so keenly that I can't put them out of my mind	①	②	③	④
39. I am a steady person	①	②	③	④
40. I get in a state of tension or turmoil as I think over my recent concerns and interests	①	②	③	④

SELF-EVALUATION QUESTIONNAIRE

Developed by C. D. Spielberger, R. L. Gorsuch and R. Lushene

STAI FORM X-1

Subject no. ..... Session ..... Phase .....

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *feel* right now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	①	②	③	④
2. I feel secure	①	②	③	④
3. I am tense	①	②	③	④
4. I am regretful	①	②	③	④
5. I feel at ease	①	②	③	④
6. I feel upset	①	②	③	④
7. I am presently worrying over possible misfortunes	①	②	③	④
8. I feel rested	①	②	③	④
9. I feel anxious	①	②	③	④
10. I feel comfortable	①	②	③	④
11. I feel self-confident	①	②	③	④
12. I feel nervous	①	②	③	④
13. I am jittery	①	②	③	④
14. I feel "high strung"	①	②	③	④
15. I am relaxed	①	②	③	④
16. I feel content	①	②	③	④
17. I am worried	①	②	③	④
18. I feel over-excited and "rattled"	①	②	③	④
19. I feel joyful	①	②	③	④
20. I feel pleasant	①	②	③	④

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How anxious did you feel during the first speech?

TOTALLY TENSE, ANXIOUS ——————————————— TOTALLY RELAXED

How anxious did you feel during the second speech?

TOTALLY TENSE, ANXIOUS ——————————————— TOTALLY RELAXED

How anxious did you feel during the third speech?

TOTALLY TENSE, ANXIOUS ——————————————— TOTALLY RELAXED

How do you feel just now?

TOTALLY TENSE, ANXIOUS ——————————————— TOTALLY RELAXED

How intoxicated or drunk do you feel?

TOTALLY SOBER (As I am when I have not drunk at all) ——————————————— TOTALLY DRUNK

MOOD CHECK LIST

Subject no. ..... Sesssion ..... Phase .....

INSTRUCTIONS: Each of the words in the following list describes feelings or mood. Please use the list to describe yours feelings at this moment, according to these instructions:

-2 stands for "I definitely do not feel ....."
-1 " " "I do not feel ....."
+1 " " "I feel slightly ....."
+2 " " "I definitely feel ....."

Dejected	-2	-1	+1	+2
Passive	-2	-1	+1	+2
Energetic	-2	-1	+1	+2
Nervous	-2	-1	+1	+2
Alert	-2	-1	+1	+2
Contented	-2	-1	+1	+2
Peaceful	-2	-1	+1	+2
Idle	-2	-1	+1	+2
Bothered	-2	-1	+1	+2
Fearful	-2	-1	+1	+2
Tired	-2	-1	+1	+2
Cheerful	-2	-1	+1	+2
Sleepy	-2	-1	+1	+2
Worried	-2	-1	+1	+2
Activated	-2	-1	+1	+2
Jittery	-2	-1	+1	+2
Drowsy	-2	-1	+1	+2
Relaxed	-2	-1	+1	+2
Vigorous	-2	-1	+1	+2
Tense	-2	-1	+1	+2
Sluggish	-2	-1	+1	+2
Uneasy	-2	-1	+1	+2
Stimulated ...	-2	-1	+1	+2
Distressed ...	-2	-1	+1	+2
Lively	-2	-1	+1	+2
Pleasant	-2	-1	+1	+2
Somnolent	-2	-1	+1	+2
Up-tight	-2	-1	+1	+2
Active	-2	-1	+1	+2
Restful	-2	-1	+1	+2
Aroused	-2	-1	+1	+2
Apprehensive .	-2	-1	+1	+2
Calm	-2	-1	+1	+2
Comfortable ..	-2	-1	+1	+2

Department of Psychology
University of Edinburgh

Study: Alcohol and Psychophysiological response

I understand that my psychophysiological response to potentially midly stressful situation will be measured on two occasions after having drunk two different doses of alcohol. These experimental situations have been fully explained to me. I am aware I will have to take a certain dose of alcohol. I understand I will receive five pounds for my participation in the two sessions the experiment involves.

Signed.....

Date.....

CALM, RELAXED	———	TENSE, ANXIOUS
HEART BEATING NORMALLY	———	HEART BEATING VERY FAST
CALM, RELAXED	———	TENSE, ANXIOUS
HEART BEATING NORMALLY	———	HEART BEATING VERY FAST
CALM, RELAXED	———	TENSE, ANXIOUS
HEART BEATING NORMALLY	———	HEART BEATING VERY FAST
CALM, RELAXED	———	TENSE, ANXIOUS
HEART BEATING NORMALLY	———	HEART BEATING VERY FAST
CALM, RELAXED	———	TENSE, ANXIOUS
HEART BEATING NORMALLY	———	HEART BEATING VERY FAST
CALM, RELAXED	———	TENSE, ANXIOUS
HEART BEATING NORMALLY	———	HEART BEATING VERY FAST

How do you feel?

TOTALLY TENSE, ANXIOUS ——————————— TOTALLY RELAXED

How intoxicated or drunk do you feel?

TOTALLY SOBER (As I am when I have not drunk at all) ——————————— TOTALLY DRUNK

Department of Psychology
University of Edinburgh

Experiment: alcohol and perception

I agree to take part in this experiment, being aware that it involves drinking a certain dose of alcohol. I confirm that (a) I do not suffer from any health problem incompatible with consuming alcohol; (b) I have not had alcohol or any kind of medication for the previous 12 hours; and (c) I have not eaten anything for the last four hours. I will not drive, ride a bicycle or handle dangerous machinery until tomorrow. I understand that I will receive 3 pounds for my participation.

Signature.....

Date.....

APPENDIX II

Computer programs used in the experiments in chapters 5, 6 and 7. These programs are original. I wrote them specifically for the purpose of conducting and analyzing the experiments in this thesis.

This program controls the recording and storing of ECG and EDA, and the presentation of auditory stimuli. It includes a low level assembly language routine to control the A/D converter allowing a sampling frequency of 50 Hz.

```
 10 REM EXPERIMENT 4
 20 *DISC
 30 PROCconstants
 40 PROCASS
 50 CLS
 60 MODE4
 70 PRINTTAB(5,9) "Press C to calibrate"
 80 PRINTTAB(5,11) "Press G to check GSR trace"
 90 PRINTTAB(5,13) "Press H to check ECG trace"
100 PRINTTAB(5,15) "Press R to record"
110 PRINTTAB(5,19) "Press E to END"
120 K$=GET$
130 IF K$="C" CLS:MODE4:PROCCALIBRATE:GOTO 60
140 IF K$="G" CLS:MODE4:PROCgsrcheck:GOTO 50
150 IF K$="H" CLS:MODE4:PROCecgcheck:GOTO 50
160 IF K$="R" CLS:MODE7:PROCrecording:GOTO 50
170 IF K$="E" CLS:END
180 GOTO 120
190 DEF PROCgsrcheck
200 PROCaxes:PROCwindows:PROCadc
210 ENDPROC
220 DEF PROCecgcheck
230 PROCaxes:PROCscaling:PROCwindows:PROCdigdisp
240 ENDPROC
250 DEF PROCconstants
260 AH%=800:AW%=1000:HM%=100:VM%=150
270 D=800/(65520DIV256)
280 MSC=&3C00:MHR=&5600:MSV=&3BFF:MGAIN=&3BFE:MVOLT=&3BFD
290 MCAL=&3BF8:DIGV1=&1005:DIGV2=&1006:PRESS=&1007
300 CLOCK=&1000:CHANNEL=&2BE
310 ?CLOCK=0:?(CLOCK+1)=0:?(CLOCK+2)=0:?(CLOCK+3)=0:?(CLOCK+4)=0
320 OSBYTE=&FFF4:OSWORD=&FFF1
330 SET=&101C:?SET=1
340 ?&FE62=3:?&FE60=1
350 FLAG=&101A:?FLAG=0
360 F=0
370 ENDPROC
380 DEF PROCaxes
390 MOVE HM%,VM%:DRAW HM%+AW%,VM%
400 MOVE HM%,VM%:DRAW HM%,VM%+AH%
410 MOVE HM%+1000,VM%:DRAW HM%+1000,VM%+800:DRAW HM%,VM%+800
420 ENDPROC
430 DEF PROCwindows
440 VDU24,HM%+4;VM%+4;HM%+AW%;VM%+AH%;
450 VDU28,0,31,39,29
460 ENDPROC
470 DEF PROCadc
480 PRINT"        Press RETURN to go to menu"
490 MOVE HM%,VM%:X=0
500 *FX17,2
510 REPEAT
520 X=X+1
530 C=ADVAL(2)DIV256
540 PROCplot
550 K$=INKEY$(0):IF ASC(K$)=13 ENDPROC
560 UNTIL X=1000
570 ENDPROC
580 DEF PROCplot
590 Y=C*D
```

```
600 Y=INT(Y)
610 DRAW HM%+X,VM%+Y
620 PRINTTAB(10,1) Y
630 ENDPROC
640 DEF PROCscaling
650 VDU5
660 @%=2
670 FOR X=0 TO 5
680 MOVE HM%+(X*200),VM%
690 PLOT 1,0,-10
700 PLOT 0,-16,-18
710 PRINT X
720 NEXT
730 MOVE HM%+1080,VM%
740 PLOT 0,0,-10
750 PLOT 0,-16,-8
760 PRINT "sec"
770 @%=10
780 VDU4
790 ENDPROC
800 DEF PROCdigdisp
810 PRINT"         Press RETURN to go to menu"
820 T=0:H%=4:N=0
830 TIME=0
840 *FX17,1
850 FOR X=1 TO 250
860 N=N+1
870 T=T+2
880 REPEAT UNTIL T=TIME
890 HR=ADVAL(1)
900 ?(MHR+N)=HR DIV256
910 K$=INKEY$(0):IF ASC(K$)=13 ENDPROC
920 NEXT
930 MOVE HM%,VM%
940 FOR X=1 TO 250
950 Y%=?(MHR+X)
960 Y%=INT(Y%*0)
970 DRAW HM%+H%,VM%+Y%
980 H%=H%+4
990 NEXT
1000 ENDPROC
1010 DEF PROCCALIBRATE
1020 PROCRATIO
1030 CLS:PRINTTAB(5,5) "A/D RATIO =";CAL
1040 INPUTTAB(5,7) "ENTER BATTERY OUTPUT = ";VOLT:?MVOLT=VOLT
1050 INPUTTAB(5,9) "ENTER GAIN = ";GAIN: ?MGAIN=GAIN
1060 F=1
1070 ENDPROC
1080 DEF PROCrecording
1090 IF F( 1 PROCCALIBRATE
1100 CLS
1110 PRINTTAB(5,10) "Press R for relax tests"
1120 PRINTTAB(5,12) "Press T for tones test"
1130 PRINTTAB(5,14) "Press P for visc. perception test"
1140 PRINTTAB(5,16) "Press S for social stress test"
1150 K$=GET$
1160 IF K$="R" CLS:PROCRELAX:ENDPROC
1170 IF K$="T" CLS:PROCTONES:ENDPROC
1180 IF K$="P" CLS:PROCPERCEPT:ENDPROC
1190 IF K$="S" CLS:PROCSOCIAL:ENDPROC
1200 GOTO 1110
1210 ENDPROC
1220 DEF PROCRATIO
1230 SADVOLT=0
1240 *FX17,3
1250 FOR X=1 TO 20
1260 TIME=0
```

```
1270 REPEAT UNTIL TIME)3
1280 ADVOLT=ADVAL(3) DIV256
1290 SADVOLT=SADVOLT+ADVOLT
1300 NEXT
1310 CAL=(SADVOLT/20)/1.035
1320 !MCAL=CAL*100
1330 ENDPROC
1340 DEF PROCOFFSET
1350 CLS
1360 INPUTTAB(5,7) "Enter supp. volt. value = ";PSV
1370 ?MSV=PSV
1380 CLS
1390 ENDPROC
1400 DEF PROCRELAX
1410 PROCCALIBRATE
1420 PROCINITIAL
1430 PROCOFFSET
1440 CLS
1450 INPUTTAB(5,10) " FIRST OR SECOND PERIOD ";relax_period
1460 IF relax_period )2 OR relax_period (1 GOTO 1440
1470 PROCSTART
1480 TIME=0
1490 CALL START%
1500 PROCrecord_relax
1510 ENDPROC
1520 DEF PROCrecord_relax
1530 IF relax_period=2 GOTO 1570
1540 *SAVE RLX1SC 3B00 5600
1550 *SAVE RLX1HR 5600 7000
1560 ENDPROC
1570 *SAVE RLX2SC 3B00 5600
1580 *SAVE RLX2HR 5600 7000
1590 ENDPROC
1600 DEF PROCASS
1610 *FX16,2
1620 *FX190,8
1630 ?CLOCK=0
1640 DIM START% 1000
1650 FOR I%=0 TO 2 STEP 2:P%=START%
1660 [ OPT I%
1670 LDA £&00:STA &70:STA &72
1680 LDA £&3C:STA &73
1690 LDA £&56:STA &71
1700 LDY £0
1710 .WAIT
1720 LDA CHANNEL
1730 CMP£2
1740 BCS WAIT
1750 .CONVERT
1760 PHA:TXA:PHA:TYA:PHA:PHP
1770 JSR SETCLOCK
1780 LDA £&80
1790 LDX £1
1800 JSR OSBYTE
1810 STY DIGV1
1820 LDA FLAG
1830 CMP £1
1840 BEQ BUTTON
1850 .WAIT2
1860 LDA CHANNEL
1870 CMP £1
1880 BEQ WAIT2
1890 LDA £&80
1900 LDX £2
1910 JSR OSBYTE
1920 STY DIGV2
```

```
1930 .TEMPO
1940 LDA £3
1950 LDX £&11
1960 LDY £&10
1970 JSR OSWORD
1980 LDA &1011
1990 CMP £1
2000 BCC TEMPO
2010 JSR SETCLOCK
2020 PLP:PLA:TAY:PLA:TAX:PLA
2030 CPY £200:BEQ SWOFF
2040 .TRANSFER
2050 LDA DIGV1
2060 STA (&70),Y
2070 LDA DIGV2
2080 STA (&72),Y
2090 INY
2100 BNE CONVERT
2110 INC &73
2120 INC &71
2130 LDA &73
2140 CMP £&40:BEQ SWON
2150 LDA &73
2160 CMP £&56
2170 BCC CONVERT
2180 .SETCLOCK
2190 LDA £0
2200 STA CLOCK
2210 LDA £4
2220 LDX £&00
2230 LDY £&10
2240 JSR OSWORD
2250 RTS
2260 JMP FIN
2270 .BUTTON
2280 LDA £0:STA DIGV2
2290 LDA &FE60:CMP £252:BCC NOPRESS
2300 LDA £1:STA DIGV2
2310 .NOPRESS
2320 JMP TEMPO
2330 .SWON:LDA SET:STA &FE60:JMP CONVERT
2340 .SWOFF:LDA £1:STA &FE60:JMP TRANSFER
2350 .FIN:RTS
2360 ] NEXT
2370 ENDPROC
2380 DEF PROCTONES
2390 PROCCALIBRATE
2400 PROCOFFSET
2410 PROCINITIAL
2420 T2=75:T3=155:T4=240:T5=320
2430 PROCSTART
2440 TIME=0:?SET=2
2450 CALL START%
2460 PRINT TIME:PRINT
2470 *SAVE SCT1 3B00 5600
2480 *SAVE HRT1 5600 7000
2490 PRINT TIME
2500 ?SET=2
2510 REPEAT UNTIL TIME/100=T2
2520 CALL START%
2530 *SAVE SCT2 3B00 5600
2540 *SAVE HRT2 5600 7000
2550 ?SET=2
2560 REPEAT UNTIL TIME/100=T3
2570 CALL START%
```

```
2580 *SAVE SCT3 3B00 5600
2590 *SAVE HRT3 5600 7000
2600 ?SET=2
2610 REPEAT UNTIL TIME/100=T4
2620 CALL START%
2630 *SAVE SCT4 3B00 5600
2640 *SAVE HRT4 5600 7000
2650 ?SET=2
2660 REPEAT UNTIL TIME/100=T5
2670 CALL START%
2680 *SAVE SCT5 3B00 5600
2690 *SAVE HRT5 5600 7000
2700 ?&FE60=1:?SET=1
2710 ENDPROC
2720 DEF PROCINITIAL
2730 *FX16,2
2740 *FX190,8
2750 ?CLOCK=0
2760 ENDPROC
2770 DEF PROCPERCEPT
2780 PROCCALIBRATE
2790 PROCINITIAL
2800 PROCOFFSET
2810 ?FLAG=1
2820 PROCSTART
2830 CALL START%
2840 *SAVE PERC1SC 3B00 5600
2850 *SAVE PERC1HR 5600 7000
2860 ?&FE62=3:?&FE60=2:TIME=0
2870 REPEAT UNTIL TIME=200
2880 ?&FE60=1
2890 CALL START%
2900 *SAVE PERC2SC 3B00 5600
2910 *SAVE PERC2HR 5600 7000
2920 ?&FE60=0:?FLAG=0
2930 ENDPROC
2940 DEF PROCSOCIAL
2950 PROCCALIBRATE
2960 PROCINITIAL
2970 PROCOFFSET
2980 PROCSTART
2990 CALL START%
3000 *SAVE SOC1SC 3B00 5600
3010 *SAVE SOC1HR 5600 7000
3020 CALL START%
3030 *SAVE SOC2SC 3B00 5600
3040 *SAVE SOC2HR 5600 7000
3050 CALL START%
3060 *SAVE SOC3SC 3B00 5600
3070 *SAVE SOC3HR 5600 7000
3080 CALL START%
3090 *SAVE SOC4SC 3B00 5600
3100 *SAVE SOC4HR 5600 7000
3110 CALL START%
3120 *SAVE SOC5SC 3B00 5600
3130 *SAVE SOC5HR 5600 7000
3140 CALL START%
3150 *SAVE SOC6SC 3B00 5600
3160 *SAVE SOC6HR 5600 7000
3170 ENDPROC
3180 DEF PROCSTART
3190 CLS:PRINTTAB(5,12) "Press RETURN to start"
3200 K$=INKEY$(0):IF ASC(K$)()13 GOTO 3200
3210 CLS:PRINTTAB(12,12) "RECORDING"
3220 ENDPROC
```

Program for automatic analysis of ECG. It includes routines to identify QRS peaks, to measure cardiac period, and to calculate heart rate second by second. The algorithm for detection of QRS peaks uses slope and amplitude as identification criteria.

```
 10 REM "STOREHR"
 20 REM ********** ECG ANALYSIS **********
 30 REM :::::::::: the program includes routines to identify
 40 REM :::::::::: QRS peaks, to measure cardiac period and
 50 REM :::::::::: to calculate HR second by second
 60 REM :::::::::: by Miguel Munoz, 1988
 70 *DISC
 80 MODE7
 90 CC=1:C=1
100 DIM HR(150)
110 DIM P(200)
120 DIM PERIOD(200)
130 P(0)=0
140 K=1
150 M=&5600:MIBI=&7000
160 THRESHOLD=30:F=0:N=1:THRES2=150
170 CLS:INPUT "ENTER DISC NUMBER";D%
180 IF D%=1 EX$="SOC"
190 IF D%=2 EX$="TON"
200 PRINT:PRINT:PRINT
210 INPUT"ENTER SUBJECT ID";ID$
220 FL$=EX$+ID$
230 *DRIVE 1
240 CH=OPENOUT FL$
250 N=1:F=0:K=1
260 IF D%=1 PROCOPEN1
270 IF D%=2 PROCOPEN2
280 FOR X=1 TO 6650
290 IF ?(M+X)<THRES2 GOTO 320
300 IF F=1 PROCPEAK:GOTO 320
310 IF ?(M+X)-?(M+X-1))THRESHOLD F=1:GOTO 320
320 NEXT
330 GOTO 420
340 DEF PROCPEAK
350 IF ?(M+X)-?(M+X-1))0 OR ?(M+X)-?(M+X-1)=0 ENDPROC
360 IF ?(M+X)-?(M+X-1)<0 P(N)=X-1:N=N+1:F=0
370 IBI=(P(N-1)-P(N-2))*10
380 PERIOD(N-1)=IBI
390 !(MIBI+K)=IBI
400 K=K+4
410 ENDPROC
420 O%=&20209
430 RE=0:P=0:HR=0:N=0:B=0
440 FOR X=1 TO K-4 STEP 4
450 IF !(MIBI+X)+RE<1000 GOTO 500
460 IF RE=0 AND !(MIBI+X))1000 GOTO 490
470 FP=(1000/!(MIBI+X))*((1000-RE)/1000):HR=(P+FP)*60
    RE=!(MIBI+X)-(1000-RE):P=(1000/!(MIBI+X))*(RE/1000):GOTO 5
480 IF RE)1000 FP=(1000/!(MIBI+X)):RE=RE-1000:P=FP*(RE/1000)
    HR=FP*60:GOTO 510
490 HR=(1000/!(MIBI+X))*60:RE=!(MIBI+X)-1000
    P=(1000/!(MIBI+X))*(RE/1000):GOTO 510
500 P=P+((1000/!(MIBI+X))*(!(MIBI+X)/1000)):RE=RE+!(MIBI+X):HR
```

```
 510 PROCSTOR
 520 IF RE(1000 GOTO 550
 530 FP=(1000/!(MIBI+X)):RE=RE-1000:P=FP*(RE/1000):HR=FP*60
 540 PROCSTOR
 550 NEXT
 560 IF CC(8 AND C(8 GOTO 250
 570 CLOSE£CH
 580 END
 590 DEF PROCOPEN2
 600 *DRIVE 0
 610 ON CC GOTO 620,640,660,680,700,720,740
 620 *LOAD HRT1
 630 GOTO 750
 640 *LOAD HRT2
 650 GOTO 750
 660 *LOAD HRT3
 670 GOTO 750
 680 *LOAD HRT4
 690 GOTO 750
 700 *LOAD HRT5
 710 GOTO 750
 720 *LOAD PERC1HR
 730 GOTO 750
 740 *LOAD PERC2HR
 750 CC=CC+1
 760 *DRIVE 1
 770 ENDPROC
 780 DEF PROCOPEN1
 790 *DRIVE 0
 800 ON C GOTO 810,830,850,870,890,910,930
 810 *LOAD RLX1HR
 820 GOTO 940
 830 *LOAD SOC1HR
 840 GOTO 940
 850 *LOAD SOC2HR
 860 GOTO 940
 870 *LOAD SOC3HR
 880 GOTO 940
 890 *LOAD SOC4HR
 900 GOTO 940
 910 *LOAD SOC5HR
 920 GOTO 940
 930 *LOAD SOC6HR
 940 C=C+1
 950 *DRIVE 1
 960 ENDPROC
 970 DEF PROCSTOR
 980 IF HR=0 ENDPROC
 990 N=N+1:HR(N)=HR
1000 IF N(2 OR N)65 ENDPROC
1010 HR=INT(HR*10)
1020 PRINT£CH,HR
1030 ENDPROC
```

Program for manual analysis of ECG trace.

```
 10 REM ECG ANALYSIS
 20 MODE 7
 30 MH=9000:M=&5600
 40 FOR X=1 TO 2999
 50 ?(MH+X)=?(M+X)
 60 NEXT
 70 D=800/(65520DIV256)
 80 G%=0
 90 MODE0
100 PROCaxes
110 PROCscaling
120 PROCwindows
130 GOTO 250
140 DEF PROCaxes
150 AH%=800:AW%=1000:HM%=100:VM%=150
160 MOVE HM%,VM%:DRAW HM%+AW%,VM%
170 MOVE HM%,VM%:DRAW HM%,VM%+AH%
180 MOVE HM%+1000,VM%:DRAW HM%+1000,VM%+800
190 DRAW HM%,VM%+800
200 ENDPROC
210 DEF PROCwindows
220 VDU24,HM%+4;VM%+4;HM%+AW%;VM%+AH%;
230 VDU28,0,31,79,29
240 ENDPROC
250 FOR S=1 TO 12
260 MOVE HM%,VM%
270 H%=4
280 FOR X=1 TO 250
290 PROCdisplay
300 NEXT
310 PROCchoice
320 G%=G%+5:MH=MH+250
330 MODE7:MODE0:PROCaxes:PROCscaling:PROCwindows
340 NEXT
350 END
360 DEF PROCdisplay
370 Y%=?(MH+X):Y%=INT(Y%*D)
380 DRAW HM%+H%,VM%+Y%
390 H%=H%+4
400 ENDPROC
410 DEF PROCchoice
420 CLS:PRINTTAB(0,1)"PRESS I FOR I.B.I. MEASURING PROCEDURE      PRE
SS C TO CONTINUE"
430 K=GET:IF K=67 GOTO 460
440 IF K=73 PROCibi
450 GOTO 430
460 CLS:CLG
470 ENDPROC
480 DEF PROCscaling
490 VDU5
500 @%=2
510 FOR X=0 TO 5
520 MOVE HM%+(X*200),VM%
530 PLOT 1,0,-10
540 PLOT 0,-16,-8
```

```
 550 PRINT X+G%
 560 NEXT
 570 MOVE HM%+1080,VM%
 580 PLOT 0,0,-10
 590 PLOT 0,-16,-8
 600 PRINT"seconds"
 610 @%=10
 620 VDU4
 630 ENDPROC
 640 DEF PROCibi
 650 X%=4:PROCdrawing
 660 K=GET
 670 IF K=46 X%=X%+4:PROCdrawing
 680 IF K=44 X%=X%-4:PROCdraw
 690 IF K=13 I1%=X%:GOTO 710
 700 GOTO 660
 710 X%=1000 - 4:PROCdrawing
 720 K=GET
 730 IF K=46 X%=X%+4:PROCdrawing
 740 IF K=44 X%=X%-4:PROCdraw
 750 IF K=13 I2%=X%:GOTO 770
 760 GOTO 720
 770 CLS:PRINTTAB(25,1)"HOW MANY PEAKS IN BETWEEN?"
 780 K=GET:IF K)49 GOTO 780
 790 IF K(48 GOTO 780
 800 M=GET:IF M)59 GOTO 800
 810 IF M(48 GOTO 800
 820 P=(M-48)+((K-48)*10)
 830 IBI=ABS(I1%-I2%)
 840 IBI=(IBI/4)*2
 850 IBI=(IBI/100)/(P+1)
 860 HR=(1/IBI)*60
 870 PROCprint
 880 ENDPROC
 890 DEF PROCdrawing
 900 MOVE HM%+X%-4,VM%
 910 PLOT 7,HM%+X%-4,VM%+799
 920 MOVE HM%+X%,VM%
 930 DRAW HM%+X%,VM%+800
 940 ENDPROC
 950DEF PROCdraw
 960 MOVE HM%+X%+4,VM%
 970 PLOT 7,HM%+X%+4,VM%+799
 980 MOVE HM%+X%,VM%
 990 DRAW HM%+X%,VM%+800
1000 ENDPROC
1010 DEF PROCprint
1020 CLS
1030 PRINT "                              I.B.I. = ";IBI;"       H.R. = ";HR
1040 PRINT"PRESS I FOR I.B.I. MEASURING                          PRESS C T
O CONTINUE"
1050 ENDPROC
```

Program to calculate skin conductance in micromhos as a function of the suppression potentials used in the recording.

```
 10 @%=&20209
 20 *DISC
 30 REM
 40 P=0
 50 TSC=0
 60 CC=1:C=1
 70 MODE7
 80 CLS:INPUT "ENTER DISC NUMBER...";D%
 90 MSC=&3C00:MSV=&3BFF:MVOLT=&3BFD:MGAIN=&3BFE:MCAL=&3BF8
100 MAVERG=&5600
110 XG=0
120 DIM SPVOLT(13)
130 DIM SPRES(13)
140 GOTO 230
150 DEF PROCSUPP
160 SPVOLT(0)=0:SPRES(1)=7.55:SPRES(2)=3.61:SPRES(3)=2.39:SPRES(4)=1.784
170 SPRES(5)=1.499:SPRES(6)=1.195:SPRES(7)=.993:SPRES(8)=.910
180 SPRES(9)=.820:SPRES(10)=.685:SPRES(11)=.622:SPRES(12)=.557
190 FOR X=1 TO 12
200 SPVOLT(X)=(?MVOLT/100)/SPRES(X)
210 NEXT
220 ENDPROC
230 PRINT:PRINT:PRINT
240 PRINT "         AVERAGING PROCEDURE"
250 PRINT:PRINT
260 PRINT "      AVERAGING EVERY DECISECOND"
270 PRINT:PRINT:PRINT
280 P=0:TSC=0
290 IF D%=1 PROCOPEN1
300 IF D%=2 PROCOPEN2
310 CAL=!MCAL/100:SV=?MSV
320 PROCSUPP
330 GOTO 380
340 PRINT:PRINT:PRINT
350 P=0:TSC=0
360 IF D%=1 PROCOPEN1
370 IF D%=2 PROCOPEN2
380 FOR X=1 TO 6600 STEP 10
390 FOR N=0 TO 9
400 IF X=1 GOTO 450
410 IF P>0 GOTO 440
420 IF ?(MSC+X+N)-?(MSC+X+N-1))40 SV=SV-1:P=5:PRINT "HERE"
430 IF ?(MSC+X+N-1)-?(MSC+X+N))40 SV=SV+1:P=5:PRINT "HERE"
440 IF P>0 P=P-1
450 V=(0.964*(1/CAL)*?(MSC+X+N))+0.028
460 V2=(V/(?MGAIN/10))+SPVOLT(SV)
470 G=1000/(?MVOLT/V2)
480 G=G*1000
490 XG=XG+G
500 NEXT
510 MXG=XG/10
520 !(MAVERG+((X DIV10)*4))=MXG
530 XG=0
540 NEXT
550 IF C<9 AND CC<7 GOTO 340
560 END
570 DEF PROCOPEN1
580 ON C GOTO 590,620,690,740,790,840,890,940.
```

```
 590 *DISC
 600 *LOAD RLX1SC
 610 C=C+1:ENDPROC
 620 *NET
 630 PRINT C
 640 *SAVE RELAX1 5600 6100
 650 *DISC
 660 *LOAD SOC1SC
 670 CAL=!MCAL/100:SV=?MSV:PROCSUPP
 680 C=C+1:ENDPROC
 690 *NET
 700 *SAVE SOC1 5600 6100
 710 *DISC
 720 *LOAD SOC2SC
 730 C=C+1:ENDPROC
 740 *NET
 750 *SAVE SOC2 5600 6100
 760 *DISC
 770 *LOAD SOC3SC
 780 C=C+1:ENDPROC
 790 *NET
 800 *SAVE SOC3 5600 6100
 810 *DISC
 820 *LOAD SOC4SC
 830 C=C+1:ENDPROC
 840 *NET
 850 *SAVE SOC4 5600 6100
 860 *DISC
 870 *LOAD SOC5SC
 880 C=C+1:ENDPROC
 890 *NET
 900 *SAVE SOC5 5600 6100
 910 *DISC
 920 *LOAD SOC6SC
 930 C=C+1:ENDPROC
 940 *NET
 950 *SAVE SOC6 5600 6100
 960 C=C+1:ENDPROC
 970 DEF PROCOPEN2
 980 ON CC GOTO 990,1020,1070,1120,1170,1220
 990 *DISC
1000 *LOAD SCT1
1010 CC=CC+1:ENDPROC
1020 *NET
1030 *SAVE SC1 5600 6100
1040 *DISC
1050 *LOAD SCT2
1060 CC=CC+1:ENDPROC
1070 *NET
1080 *SAVE SC2 5600 6100
1090 *DISC
1100 *LOAD SCT3
1110 CC=CC+1:ENDPROC
1120 *NET
1130 *SAVE SC3 5600 6100
1140 *DISC
1150 *LOAD SCT4
1160 CC=CC+1:ENDPROC
1170 *NET
1180 *SAVE SC4 5600 6100
1190 *DISC
1200 *LOAD SCT5
1210 CC=CC+1:ENDPROC
1220 *NET
1230 *SAVE SC5 5600 6100
1240 CC=CC+1:ENDPROC
```

APPENDIX III

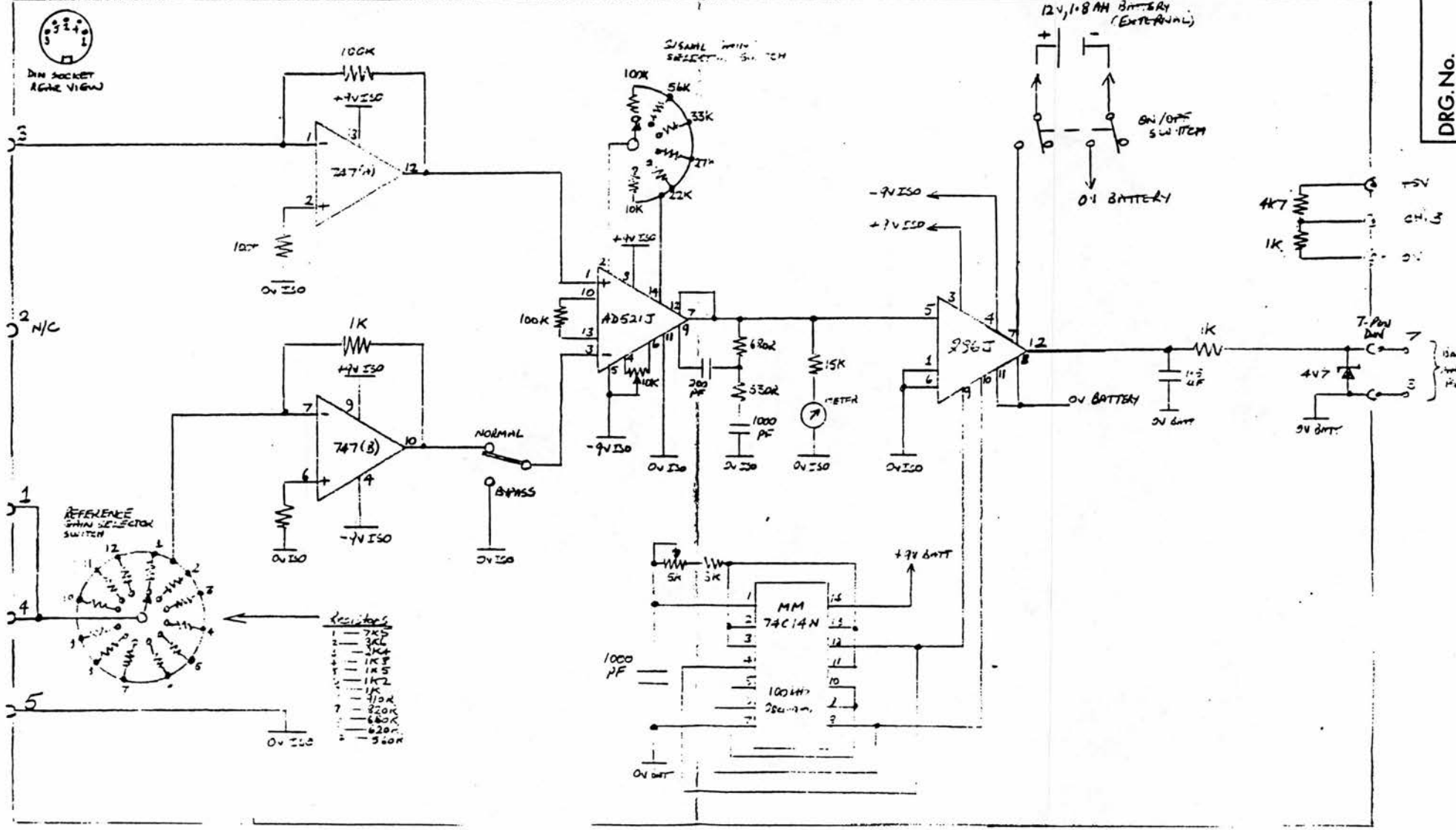
Circuit diagram of EDA amplifier. This transducer-amplifier is based on the active circuit method outlined by Lowry (1977). I adapted Lowry's circuit. I designed the step-by-step suppression voltage system, and I made all the calculations of the resistor values and amplifier settings in order to achieve the required resolution within the input range of the A/D converter of the BBC microcomputer. The electronic workshop in the Department of Psychology (Univ. of Edinburgh) designed the optical isolation circuit.

PART OF

DO NOT SCALE

SHEET OF

DRG. No.



ISSUE	DESCRIPTION	APPD.	DATE

TITLE

GSR ISOLATION AMPLIFIER.

DRAWN	TRACED	CHECKED	APPROVED	DATE

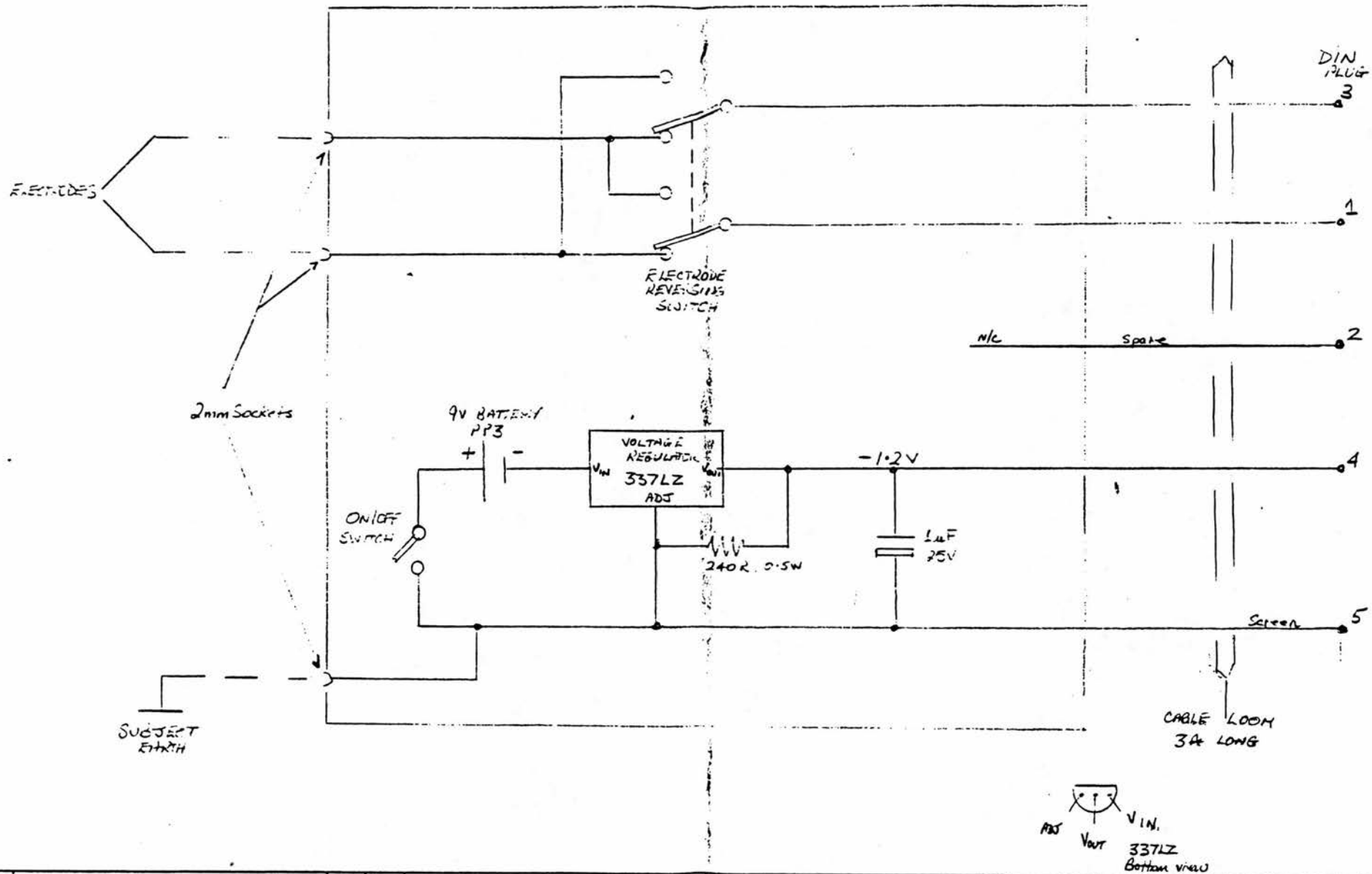
DRAWING No.

PART OF

DO NOT SCALE

SHEET OF

DRG. No.



ISSUE	DESCRIPTION	APPD.	DATE

TITLE

GSR ELECTRODES

STABILISED VOLTAGE BOX

DRAWN	TRACED	CHECKED	APPROVED	DATE
PB				9/9/88

DRAWING No.

APPENDIX IV

EDA: Electrodermal Activity. This term is preferred today to GSR (Galvanic Skin Response) used by earlier investigators.

SCR: Skin Conductance Response. It refers to momentary fluctuations in skin conductance.

SCL: Skin Conductance Level refers to the baseline skin response at any given time.

NS-SCR: Non Specific Skin Conductance Response. It refers to the SCR that cannot be attributed to specific stimuli.

QRS complex: It is a wave caused by spread of excitation through the muscle of the ventricles. The peak of the QRS complex is the point of highest voltage in the cardiac cycle.